

Connecting via Winsock to STN

STN

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PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * Welcome to STN International * * * * *

NEWS 1 Web Page for STN Seminar Schedule - N. America
NEWS 2 MAY 01 New CAS web site launched
NEWS 3 MAY 08 CA/CAPLUS Indian patent publication number format defined
NEWS 4 MAY 14 RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS 5 MAY 21 BIOSIS reloaded and enhanced with archival data
NEWS 6 MAY 21 TOXCENTER enhanced with BIOSIS reload
NEWS 7 MAY 21 CA/CAPLUS enhanced with additional kind codes for German patents
NEWS 8 MAY 22 CA/CAPLUS enhanced with IPC reclassification in Japanese patents
NEWS 9 JUN 27 CA/CAPLUS enhanced with pre-1967 CAS Registry Numbers
NEWS 10 JUN 29 STN Viewer now available
NEWS 11 JUN 29 STN Express, Version 8.2, now available
NEWS 12 JUL 02 LEMBASE coverage updated
NEWS 13 JUL 02 LMEDLINE coverage updated
NEWS 14 JUL 02 SCISEARCH enhanced with complete author names
NEWS 15 JUL 02 CHEMCATS accession numbers revised
NEWS 16 JUL 02 CA/CAPLUS enhanced with utility model patents from China
NEWS 17 JUL 16 CAPLUS enhanced with French and German abstracts
NEWS 18 JUL 18 CA/CAPLUS patent coverage enhanced
NEWS 19 JUL 26 USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS 20 JUL 30 USGENE now available on STN
NEWS 21 AUG 06 CAS REGISTRY enhanced with new experimental property tags
NEWS 22 AUG 06 BEILSTEIN updated with new compounds
NEWS 23 AUG 06 FSTA enhanced with new thesaurus edition
NEWS 24 AUG 13 CA/CAPLUS enhanced with additional kind codes for granted patents
NEWS 25 AUG 20 CA/CAPLUS enhanced with CAS indexing in pre-1907 records

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

NEWS HOURS STN Operating Hours Plus Help Desk Availability
NEWS LOGIN Welcome Banner and News Items
NEWS IPC8 For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

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result in loss of user privileges and other penalties.

* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 12:45:17 ON 24 AUG 2007

=> fil reg

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

FILE 'REGISTRY' ENTERED AT 12:45:23 ON 24 AUG 2007

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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 23 AUG 2007 HIGHEST RN 945525-31-5

DICTIONARY FILE UPDATES: 23 AUG 2007 HIGHEST RN 945525-31-5

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 29, 2007

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stngen/stndoc/properties.html>

=> e eicosapentanoic acid/cn

E1	1	EICOSAPENTAENOYL CHLORIDE/CN
E2	1	EICOSAPENTAENOYL CHLORIDE, (ALL-Z) -/CN
E3	0 -->	EICOSAPENTANOIC ASID/CN
E4	1	EICOSAPENTAYNOIC ACID/CN
E5	1	EICOSAPHINGA-11-ENINE/CN
E6	1	EICOSAPHINGENINE/CN
E7	1	EICOSAPHOSPHA(5) FULLERANE-C20-IH/CN
E8	1	EICOSAPHOSPHA(5) FULLERANE-C20-IH, COMPD. WITH SODIUM (1:1)/CN
E9	1	EICOSAPHOSPHA(5) FULLERANE-C20-IH, COMPD. WITH SODIUM (1:1), ION(1+)/CN
E10	1	EICOSAPHOSPHA(5) FULLERANE-C20-IH, EICOSAOXIDE/CN
E11	1	EICOSAPHOSPHA(5) FULLERANE-C20-IH, EICOSAOXIDE, COMPD. WITH SODIUM (1:1)/CN
E12	1	EICOSAPHOSPHA(5) FULLERANE-C20-IH, EICOSAOXIDE, COMPD. WITH SODIUM ION (NA1-) (1:1)/CN

=> s e4

L1 1 "EICOSAPENTAYNOIC ACID"/CN

=> d

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
RN 121139-92-2 REGISTRY

ED Entered STN: 16 Jun 1989
CN Eicosapentaynoic acid (9CI) (CA INDEX NAME)
MF C20 H20 O2
CI IDS
SR CA
LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER

CM 1

CRN 506-30-9
CMF C20 H40 O2

HO2C (CH2)18 Me

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> sel chen 11

'CHEN' IS NOT A VALID FIELD CODE FOR FILE 'REGISTRY'

The following are valid field codes:

AF ----- Alternate Molecular Formula
AR ----- Alternate Registry Number
CCI ----- Component Substance Class Identifier
CHEM ---- CAS Registry Numbers and Selected Names
CI ----- Substance Class Identifier
CMF ----- Component Molecular Formulas
CN ----- Chemical Names (Up to 50)
CRN ----- Component Registry Numbers
DEF ----- Definition
DR ----- Deleted Registry Number
EA ----- Elemental Analysis for Ring System
ES ----- Elemental Sequence for Ring System
FCN ----- All Chemical Names
FS ----- File Segment
IN ----- CA Index Name
LC ----- CAS Registry Number Locator
MF ----- Molecular Formula
NAME ---- Selected Substance Names
PCT ----- Polymer Class Term
PR ----- Preferred Registry Number
PN ----- Patent Number
RF ----- Ring System Formula
RID ----- Ring Identifier
RL ----- CAlus Super roles
RLD ----- CAlus Super roles for non-specific Derivatives
RL.NP --- Roles from Non-patents
RL.P --- Roles from Patents
RLD.NP -- Roles for non-specific derivatives from Non-patents
RLD.P --- Roles for non-specific derivatives from Patents
RLS ----- All CAlus Super roles
RLS.NP -- CAlus Super roles - Non-patents
RLS.P --- CAlus Super roles - Patents
RN ----- CAS Registry Number
RR ----- Replacing Registry Number
SCN ----- Short Chemical Name (IN and OTHER NAMES)
SEQ ----- Protein Sequence Display using 1 Letter Amino Acid Codes (default)
SEQ3 ----- Protein Sequence Display using 3 Letter Amino Acid Codes
SQEFP --- Protein Sequence (exact family search form)

SQEN ---- Nucleic Acid Sequence (exact search form)
 SQEP ---- Protein Sequence (exact search form)
 SQSFP --- Protein Sequence (subsequence family search form)
 SQSN ---- Nucleic Acid Sequence (subsequence search form)
 SQSP ---- Protein Sequence (subsequence search form)
 SR ----- Source of Registration
 SZ ----- Size for Ring System
 TAGS ---- Experimental Tags (same as ETAG)
 ENTER DISPLAY CODE (CHEM) OR ? :end

=> sel chem l1
 E1 THROUGH E2 ASSIGNED

=> d

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
 RN 121139-92-2 REGISTRY
 ED Entered STN: 16 Jun 1989
 CN Eicosapentaynoic acid (9CI) (CA INDEX NAME)
 MF C20 H20 O2
 CI IDS
 SR CA
 LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER

CM 1

CRN 506-30-9
 CMF C20 H40 O2

HO₂C⁻ (CH₂)₁₈-Me

1 REFERENCES IN FILE CA (1907 TO DATE)
 1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> d his

(FILE 'HOME' ENTERED AT 12:45:17 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 12:45:23 ON 24 AUG 2007

E EICOSAPENTANOIC ACID/CN

L1 1 S: E4
 SEL CHEM L1

=> e eicosapentanoic acid/cn

E1 1 EICOSAPENTAENOYL CHLORIDE/CN
 E2 1 EICOSAPENTAENOYL CHLORIDE, (ALL-Z)-/CN
 E3 0 --> EICOSAPENTANOIC ACID/CN
 E4 1 EICOSAPENTAYNOIC ACID/CN
 E5 1 EICOSAPHINGA-11-ENINE/CN
 E6 1 EICOSAPHINGENINE/CN
 E7 1 EICOSAPHOSPHA(5) FULLERANE-C20-IH/CN
 E8 1 EICOSAPHOSPHA(5) FULLERANE-C20-IH, COMPD. WITH SODIUM (1:1)/C
 N
 E9 1 EICOSAPHOSPHA(5) FULLERANE-C20-IH, COMPD. WITH SODIUM (1:1),
 ION(1+)/CN
 E10 1 EICOSAPHOSPHA(5) FULLERANE-C20-IH, EICOSAOXIDE/CN
 E11 1 EICOSAPHOSPHA(5) FULLERANE-C20-IH, EICOSAOXIDE, COMPD. WITH S
 ODIUM (1:1)/CN

E12 1 EICOSAPHOSPHA(5)FULLERANE-C20-IH, EICOSAOXIDE, COMPD. WITH S
ODIUM ION (Na1-) (1:1)/CN

=> fil BIOSIS, CA, CAPLUS
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
15.49	15.70

FULL ESTIMATED COST

FILE 'BIOSIS' ENTERED AT 12:53:32 ON 24 AUG 2007
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=> BIOSIS, uspatfull, CAPLUS
BIOSIS, IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> file BIOSIS, uspatfull, CAPLUS
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
1.81	17.51

FULL ESTIMATED COST

FILE 'BIOSIS' ENTERED AT 12:53:52 ON 24 AUG 2007
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FILE 'USPATFULL' ENTERED AT 12:53:52 ON 24 AUG 2007
CA INDEXING COPYRIGHT (C) 2007 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'CAPLUS' ENTERED AT 12:53:52 ON 24 AUG 2007
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=> s l1
L2 2 L1
=> d his

(FILE 'HOME' ENTERED AT 12:45:17 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 12:45:23 ON 24 AUG 2007
E EICOSAPENTANOIC ASID/CN
L1 1 S E4
SEL CHEM L1
E EICOSAPENTANOIC ACID/CN

FILE 'BIOSIS, CA, CAPLUS' ENTERED AT 12:53:32 ON 24 AUG 2007

FILE 'BIOSIS, USPATFULL, CAPLUS' ENTERED AT 12:53:52 ON 24 AUG 2007

L2 2 S L1

=> s EICOSAPENTANOIC ACID
L3 916 EICOSAPENTANOIC ACID

=> s EICOSAPENTAYNOIC ACID
L4 13 EICOSAPENTAYNOIC ACID

=> s 121139-92-2/rn
'RN' IS NOT A VALID FIELD CODE
L5 1 121139-92-2/RN

=> s l3 not l4
L6 915 L3 NOT L4

=> s l3 or l4
L7 928 L3 OR L4

=> s l7 anorexia
MISSING OPERATOR L7 ANOREXIA
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l7 and anorexia
L8 30 L7 AND ANOREXIA

=> s l7 and bulimia
L9 3 L7 AND BULIMIA

=> s l9 or l8
L10 30 L9 OR L8

=> s l9 and py<2003
L11 2 L9 AND PY<2003

=> d scan

L11 2 ANSWERS USPATFULL
AN 2002:70000 USPATFULL
TI Anticachectic composition
NCL NCLM: 514/342.000
NCLS: 514/340.000; 514/369.000; 514/376.000; 514/389.000
IC [7]
ICM A61K031-44
ICS A61K031-425; A61K031-42; A61K031-415
IPCI A61K0031-44 [ICM,7]; A61K0031-425 [ICS,7]; A61K0031-42 [ICS,7];
A61K0031-415 [ICS,7]
IPCR C07D0277-20 [I,A]; A61K0031-42 [I,C*]; A61K0031-42 [I,A];
A61K0031-421 [I,C*]; A61K0031-421 [I,A]; A61K0031-422 [I,C*];
A61K0031-422 [I,A]; A61K0031-425 [I,C*]; A61K0031-425 [I,A];
A61K0031-426 [I,C*]; A61K0031-426 [I,A]; A61K0031-427 [I,C*];
A61K0031-427 [I,A]; A61K0031-44 [I,C*]; A61K0031-44 [I,A];
A61K0031-4427 [I,C*]; A61K0031-4439 [I,A]; A61P0003-00 [I,C*];
A61P0003-00 [I,A]; A61P0003-08 [I,A]; A61P0003-10 [I,A];
A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07D0263-00 [I,C*];
C07D0263-44 [I,A]; C07D0277-00 [I,C*]; C07D0277-34 [I,A];
C07D0413-00 [I,C*]; C07D0413-06 [I,A]; C07D0413-14 [I,A];
C07D0417-00 [I,C*]; C07D0417-06 [I,A]; C07D0417-12 [I,A];
C07D0417-14 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):2

L11 2 ANSWERS USPATFULL
 AN 2000:113975 USPATFULL
 TI Anticachectic composition
 NCL NCLM: 514/343.000
 NCLS: 514/369.000; 514/376.000; 514/425.000
 IC [7]
 ICM A61K031-44
 ICS A61K031-42; A61K031-425; A61K031-40
 IPCI A61K0031-44 [ICM,7]; A61K0031-42 [ICS,7]; A61K0031-425 [ICS,7];
 A61K0031-40 [ICS,7]
 IPCR A61K0031-422 [I,A]; A61K0031-422 [I,C*]; A61K0031-427 [I,A];
 A61K0031-427 [I,C*]; A61K0031-4427 [I,C*]; A61K0031-4439 [I,A]
 PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

ALL ANSWERS HAVE BEEN SCANNED

=> d l11 1-2 abs ibib hit

L11 ANSWER 1 OF 2 USPATFULL on STN
 AB A medicinal composition for the prophylaxis and treatment of cachexia
 which comprises a compound of the formula: ##STR1##

 wherein R represents a hydrocarbon group that may be substituted or a
 heterocyclic group that may be substituted; Y represents a group of the
 formula --CO--, --CH(OH)--, or --NR.sup.3-- (R.sup.3 represents an alkyl
 group that may be substituted); m is 0 or 1; n is 0, 1 or 2; X
 represents CH or N; A represents a bond or a bivalent aliphatic
 hydrocarbon group having 1 to 7 carbon atoms; Q represents oxygen or
 sulfur; R.sup.1 represents hydrogen or an alkyl group; ring E may have
 further 1 to 4 substituents, which may form a ring in combination with
 R.sup.1; L and M respectively represent hydrogen or may be combined with
 each other to form a bond, provided that when m and n are 0, X
 represents CH, A represents a bond, Q represents sulfur, R.sup.1, L and
 M respectively represent hydrogen, and ring E does not have further
 substituents, R does not represent dihydrobenzopyranyl; or a salt
 thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:70000 USPATFULL
 TITLE: Anticachectic composition
 INVENTOR(S): Momose, Yu, Takarazuka, JAPAN
 Matsutani, Etsuya, Suita, JAPAN
 Sohda, Takashi, Takatsuki, JAPAN
 PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, JAPAN
 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6365607	B1	20020402
APPLICATION INFO.:	US 2000-605628		20000628 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 155593, now patented, Pat. No. US 6110948		

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1996-82845	19960404
	JP 1997-27957	19970212
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Goldberg, Jerome D.	

LEGAL REPRESENTATIVE: Wenderoth, Lind & Ponack, L.L.P.
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 829

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 6365607 B1 20020402 <--

SUMM Furthermore, drugs which are documented as being anticachectic in an animal model or clinically, such as cyclooxygenase inhibitors (e.g. indomethacin) [Cancer Research, 49, 5935-5939, (1989)], progesterone derivatives (e.g. megestrol acetate) [Journal of Clinical Oncology, 12, 213-225, 1994], glucocorticoids (e.g. dexamethasone), metoclopramides, tetrahydrocannabinols (the same literature as above), lipid metabolism improving agents (e.g. eicosapentanoic acid) [British Journal of Cancer, 68, 314-318, 1993], growth hormone, IGF-1, and antibodies to the cachexia-inducing factors TNF- α , LIF, IL-6, and oncostatin M may also be used together with the composition for prophylaxis and treatment of the present invention.

SUMM Among the compound of the present invention, especially a compound which has a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms for A in the formula (I) or a salt thereof, has an activity to prevent and treat atherosclerosis, and an activity to regulate appetite and food intake in disorders associated with under-eating, such as anorexia nervosa, and disorders associated with over-eating, such as obesity and anorexia bulimia. Therefore, such a compound or a salt thereof can be used, as such or by providing in a pharmaceutic dosage form in the same manner as described above, as an agent for prophylaxis and treatment of atherosclerosis, or medicine for the regulation of appetite and food intake.

L11 ANSWER 2 OF 2 USPATFULL on STN

AB A medicinal composition for the prophylaxis and treatment of cachexia which comprises a compound of the formula: ##STR1## wherein R represents a hydrocarbon group that may be substituted or a heterocyclic group that may be substituted; Y represents a group of the formula --CO--, --CH(OH)--, or --NR^{sup.3} -- (R^{sup.3} represents an alkyl group that may be substituted); m is 0 or 1; n is 0, 1 or 2; X represents CH or N; A represents a bond or a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms; Q represents oxygen or sulfur; R^{sup.1} represents hydrogen or an alkyl group; ring E may have further 1 to 4 substituents, which may form a ring in combination with R^{sup.1}; L and M respectively represent hydrogen or may be combined with each other to form a bond, provided that when m and n are 0, X represents CH, A represents a bond, Q represents sulfur, R^{sup.1}, L and M respectively represent hydrogen, and ring E does not have further substituents, R does not represent dihydrobenzopyranyl; or a salt thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2000:113975 USPATFULL

TITLE: Anticachectic composition

INVENTOR(S): Momose, Yu, Takarazuka, Japan
Matsutani, Etsuya, Suita, Japan
Sohda, Takashi, Takatsuki, Japan

PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, Japan
(non-U.S. corporation)

NUMBER	KIND	DATE
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PATENT INFORMATION:	US 6110948	20000829
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APPLICATION INFO.: WO 9737656 19971016 <--
 US 1998-155593 19980930 (9)
 WO 1997-JP1148 19970403
 19980930 PCT 371 date
 19980930 PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1996-82845	19960404
	JP 1997-27957	19970212
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Goldberg, Jerome D.	
LEGAL REPRESENTATIVE:	Wenderoth, Lind & Ponack, LLP.	
NUMBER OF CLAIMS:	11	
EXEMPLARY CLAIM:	1	
LINE COUNT:	779	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 6110948 20000829 <--
 WO 9737656 19971016 <--

SUMM Furthermore, drugs which are documented as being anticachectic in an animal model or clinically, such as cyclooxygenase inhibitors (e.g. indomethacin) [Cancer Research, 49, 5935-5939, (1989)], progesterone derivatives (e.g. megestrol acetate) [Journal of Clinical Oncology, 12, 213-225, 1994], glucocorticoids (e.g. dexamethasone), metoclopramides, tetrahydrocannabinols (the same literature as above), lipid metabolism improving agents (e.g. eicosapentanoic acid) [British Journal of Cancer, 68, 314-318, 1993], growth hormone, IGF-1, and antibodies to the cachexia-inducing factors TNF- α , LIF, IL-6, and oncostatin M may also be used together with the composition for prophylaxis and treatment of the present invention.

SUMM Among the compound of the present invention, especially a compound which has a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms for A in the formula (I) or a salt thereof, has an activity to prevent and treat atherosclerosis, and an activity to regulate appetite and food intake in disorders associated with under-eating, such as anorexia nervosa, and disorders associated with over-eating, such as obesity and anorexia bulimia. Therefore, such a compound or a salt thereof can be used, as such or by providing in a pharmaceutic dosage form in the same manner as described above, as an agent for prophylaxis and treatment of atherosclerosis, or medicine for the regulation of appetite and food intake.

=> d his

(FILE 'HOME' ENTERED AT 12:45:17 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 12:45:23 ON 24 AUG 2007

E EICOSAPENTANOIC ASID/CN
 L1 1 S E4
 SEL CHEM L1
 E EICOSAPENTANOIC ACID/CN

FILE 'BIOSIS, CA, CAPLUS' ENTERED AT 12:53:32 ON 24 AUG 2007

FILE 'BIOSIS, USPATFULL, CAPLUS' ENTERED AT 12:53:52 ON 24 AUG 2007

L2 2 S L1
 L3 916 S EICOSAPENTANOIC ACID

L4 13 S EICOSAPENTAYNOIC ACID
L5 1 S 121139-92-2/RN
L6 915 S L3 NOT L4
L7 928 S L3 OR L4
L8 30 S L7 AND ANOREXIA
L9 3 S L7 AND BULIMIA
L10 30 S L9 OR L8
L11 2 S L9 AND PY<2003

=> s l3 or l4 or l5

L12 928 L3 OR L4 OR L5

=> s l10 and py<2003

L13 11 L10 AND PY<2003

=> d scan

L13 11 ANSWERS USPATFULL
AN 2001:97948 USPATFULL
TI Oxyiminoalkanoic acid derivatives with hypoglycemic and hypolipidemic activity
NCL NCLM: 514/364.000
NCLS: 514/365.000; 514/372.000; 514/374.000; 514/378.000; 548/131.000; 548/143.000; 548/204.000; 548/214.000; 548/235.000; 548/236.000; 548/247.000; 548/248.000
IC [7]
ICM A61K031-4245
ICS A61K031-421; C07D003-06; A61P029-00
IPCI A61K0031-4245 [ICM,7]; A61K0031-421 [ICS,7]; C07D0003-06 [ICS,7]; A61P0029-00 [ICS,7]
IPCR C07D0213-00 [I,C*]; C07D0213-74 [I,A]; C07D0215-00 [I,C*]; C07D0215-14 [I,A]; C07D0239-00 [I,C*]; C07D0239-42 [I,A]; C07D0261-00 [I,C*]; C07D0261-08 [I,A]; C07D0263-00 [I,C*]; C07D0263-32 [I,A]; C07D0271-00 [I,C*]; C07D0271-06 [I,A]; C07D0277-00 [I,C*]; C07D0277-24 [I,A]; C07D0413-00 [I,C*]; C07D0413-12 [I,A]; C07D0471-00 [I,C*]; C07D0471-04 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):10

L13 11 ANSWERS BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Metabolic responses to tumour disease and progression: Tumour-host interaction.
IT Miscellaneous Descriptors
cachexia; carbohydrate metabolism; fat metabolism; metabolic rate; metabolic responses; protein metabolism; tumor-host interaction.

L13 11 ANSWERS USPATFULL
AN 95:29628 USPATFULL
TI Nutritional product for persons infected with human immunodeficiency virus
NCL NCLM: 514/021.000
NCLS: 426/656.000; 426/800.000; 514/002.000; 514/023.000
IC [6]
ICM A16K037-02
ICS A16K031-70; A16K035-60
IPCI A16K0037-02 [ICM,6]; A16K0031-70 [ICS,6]; A16K0035-60 [ICS,6]
IPCR A23L0001-29 [I,C*]; A23L0001-29 [I,A]; A23L0001-305 [I,C*]; A23L0001-305 [I,A]; A61K0038-01 [I,C*]; A61K0038-01 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L13 11 ANSWERS USPATFULL
AN 2001:112384 USPATFULL
TI Methods of treating immunopathologies using polyunsaturated fatty acids
NCL NCLM: 514/560.000
NCLS: 514/627.000; 514/826.000; 514/861.000; 514/863.000; 514/885.000;
514/903.000

IC [7]
ICM A61K031-20
IPCI A61K0031-20 [ICM,7]; A61K0031-185 [ICM,7,C*]
IPCR A61K0031-185 [I,C*]; A61K0031-202 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L13 11 ANSWERS BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Cachexia related to mechanisms and nutrients.
IT Miscellaneous Descriptors
body weight loss; lean body mass; ubiquitin-proteasome proteolytic
pathway; Meeting Abstract

L13 11 ANSWERS USPATFULL
AN 96:1451 USPATFULL
TI Method of providing enternal nutritional support to persons infected
with human immunodeficiency virus
NCL NCLM: 514/021.000
NCLS: 426/641.000; 426/648.000; 426/654.000; 426/656.000; 426/657.000

IC [6]
ICM A23J003-16
ICS A23L001-052; A61K038-17; A61K047-42
IPCI A23J0003-16 [ICM,6]; A23J0003-00 [ICM,6,C*]; A23L0001-052
[ICS,6]; A61K0038-17 [ICS,6]; A61K0047-42 [ICS,6]
IPCR A23L0001-29 [I,C*]; A23L0001-29 [I,A]; A23L0001-305 [I,C*];
A23L0001-305 [I,A]; A61K0038-01 [I,C*]; A61K0038-01 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L13 11 ANSWERS USPATFULL
AN 2002:70000 USPATFULL
TI Anticachectic composition
NCL NCLM: 514/342.000
NCLS: 514/340.000; 514/369.000; 514/376.000; 514/389.000

IC [7]
ICM A61K031-44
ICS A61K031-425; A61K031-42; A61K031-415
IPCI A61K0031-44 [ICM,7]; A61K0031-425 [ICS,7]; A61K0031-42 [ICS,7];
A61K0031-415 [ICS,7]
IPCR C07D0277-20 [I,A]; A61K0031-42 [I,C*]; A61K0031-42 [I,A];
A61K0031-421 [I,C*]; A61K0031-421 [I,A]; A61K0031-422 [I,C*];
A61K0031-422 [I,A]; A61K0031-425 [I,C*]; A61K0031-425 [I,A];
A61K0031-426 [I,C*]; A61K0031-426 [I,A]; A61K0031-427 [I,C*];
A61K0031-427 [I,A]; A61K0031-44 [I,C*]; A61K0031-44 [I,A];
A61K0031-4427 [I,C*]; A61K0031-4439 [I,A]; A61P0003-00 [I,C*];
A61P0003-00 [I,A]; A61P0003-08 [I,A]; A61P0003-10 [I,A];
A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07D0263-00 [I,C*];
C07D0263-44 [I,A]; C07D0277-00 [I,C*]; C07D0277-34 [I,A];
C07D0413-00 [I,C*]; C07D0413-06 [I,A]; C07D0413-14 [I,A];
C07D0417-00 [I,C*]; C07D0417-06 [I,A]; C07D0417-12 [I,A];
C07D0417-14 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L13 11 ANSWERS BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Tumour anorexia - tumour cachexia in case of gastrointestinal
tumours: Standards and visions
Original Title: Tumoranorexie - Tumorkachexie bei gastrointestinalen

Tumoren: Standards und Visionen..

L13 11 ANSWERS USPATFULL
AN 2000:113975 USPATFULL
TI Anticachectic composition
NCL NCLM: 514/343.000
NCLS: 514/369.000; 514/376.000; 514/425.000
IC [7]
ICM A61K031-44
ICS A61K031-42; A61K031-425; A61K031-40
IPCI A61K0031-44 [ICM,7]; A61K0031-42 [ICS,7]; A61K0031-425 [ICS,7];
A61K0031-40 [ICS,7]
IPCR A61K0031-422 [I,A]; A61K0031-422 [I,C*]; A61K0031-427 [I,A];
A61K0031-427 [I,C*]; A61K0031-4427 [I,C*]; A61K0031-4439 [I,A]
PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L13 11 ANSWERS USPATFULL
AN 2002:332756 USPATFULL
TI Oxyiminoalkanoic acid derivatives
NCL NCLM: 514/365.000
NCLS: 514/370.000; 514/372.000; 548/194.000; 548/200.000; 548/204.000;
548/214.000
IC [7]
ICM A61K031-425
ICS A61K031-426; C07D275-02; C07D277-24
IPCI A61K0031-425 [ICM,7]; A61K0031-426 [ICS,7]; C07D0275-02 [ICS,7];
C07D0275-00 [ICS,7,C*]; C07D0277-24 [ICS,7]; C07D0277-00
[ICS,7,C*]
IPCR A61K0031-185 [I,C*]; A61K0031-195 [I,A]; A61K0031-21 [I,C*];
A61K0031-235 [I,A]; A61K0031-421 [I,C*]; A61K0031-421 [I,A];
A61K0031-422 [I,C*]; A61K0031-422 [I,A]; A61K0031-4245 [I,C*];
A61K0031-4245 [I,A]; A61K0031-426 [I,C*]; A61K0031-426 [I,A];
A61K0031-4353 [I,C*]; A61K0031-437 [I,A]; A61K0031-44 [I,C*];
A61K0031-44 [I,A]; A61K0031-4402 [I,C*]; A61K0031-4402 [I,A];
A61K0031-4427 [I,C*]; A61K0031-4439 [I,A]; A61K0031-47 [I,C*];
A61K0031-47 [I,A]; A61K0031-496 [I,C*]; A61K0031-496 [I,A];
A61K0031-505 [I,C*]; A61K0031-505 [I,A]; A61P0003-00 [I,C*];
A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0009-00 [I,C*];
A61P0009-10 [I,A]; A61P0029-00 [I,C*]; A61P0029-00 [I,A];
A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07D0213-00 [I,C*];
C07D0213-74 [I,A]; C07D0215-00 [I,C*]; C07D0215-14 [I,A];
C07D0239-00 [I,C*]; C07D0239-42 [I,A]; C07D0261-00 [I,C*];
C07D0261-08 [I,A]; C07D0263-00 [I,C*]; C07D0263-32 [I,A];
C07D0271-00 [I,C*]; C07D0271-06 [I,A]; C07D0277-00 [I,C*];
C07D0277-24 [I,A]; C07D0413-00 [I,C*]; C07D0413-04 [I,A];
C07D0413-12 [I,A]; C07D0471-00 [I,C*]; C07D0471-04 [I,A]
PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L13 11 ANSWERS USPATFULL
AN 94:62434 USPATFULL
TI Method of impeding apoptosis of CD4 cells in persons infected with human
immunodeficiency virus.
NCL NCLM: 514/002.000
NCLS: 426/044.000; 426/046.000; 426/419.000; 426/656.000; 426/658.000;
426/800.000; 514/021.000; 530/378.000
IC [5]
ICM A61K037-02
IPCI A61K0037-02 [ICM,5]
IPCR A23L0001-29 [I,C*]; A23L0001-29 [I,A]; A61K0038-01 [I,C*];
A61K0038-01 [I,A]; A61K0045-00 [I,C*]; A61K0045-06 [I,A]
PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

ALL ANSWERS HAVE BEEN SCANNED

=> d 113 1-11 abs ibib hit kwic

L13 ANSWER 1 OF 11 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AB The development of progressive malnutrition or cachexia is frequent in patients with gastrointestinal cancer - especially in patients with a carcinoma of the pancreas. The cachexia syndrome which is characterised by loss of body weight, negative nitrogen balance and fatigue significantly affects patients' quality of life, morbidity and survival. Because the currently established therapeutical strategies are often disappointing many physicians tended to develop a therapeutical nihilism. Cancer anorexia and cachexia are two distinct syndromes which may have synergistic effects in a patient. This review highlights the growing understanding of the multidimensional pathophysiological background. An algorithm of the current treatment strategies is given. In addition, we discuss new anabolic and anticatabolic agents (e.g. eicosapentanoic acid) and the results from first clinical trials.

ACCESSION NUMBER: 2003:47462 BIOSIS

DOCUMENT NUMBER: PREV200300047462

TITLE: Tumour anorexia - tumour cachexia in case of gastrointestinal tumours: Standards and visions.
Original Title: Tumoranorexie - Tumorkachexie bei gastrointestinalen Tumoren: Standards und Visionen..

AUTHOR(S): Ockenga, J. [Reprint Author]; Pirlich, M.; Gastell, S.; Lochs, H.

CORPORATE SOURCE: Medizinische Klinik mit Schwerpunkt Gastroenterologie, Hepatologie und Endokrinologie, Humboldt Universitaet, Charite, Schumannstrasse 20/21, 10117, Berlin, Germany
johann.ockenga@charite.de

SOURCE: Zeitschrift fuer Gastroenterologie, (November 2002) Vol. 40, No. 11, pp. 929-936. print.
CODEN: ZGASAX. ISSN: 0044-2771.

DOCUMENT TYPE: Article

LANGUAGE: German

ENTRY DATE: Entered STN: 15 Jan 2003
Last Updated on STN: 15 Jan 2003

TI Tumour anorexia - tumour cachexia in case of gastrointestinal tumours: Standards and visions.
Original Title: Tumoranorexie - Tumorkachexie bei gastrointestinalen Tumoren: Standards und Visionen..

SO Zeitschrift fuer Gastroenterologie, (November 2002) Vol. 40, No. 11, pp. 929-936. print.
CODEN: ZGASAX. ISSN: 0044-2771.

AB The development of progressive malnutrition or cachexia is frequent in patients with gastrointestinal cancer - especially in patients with a carcinoma of the pancreas. The cachexia syndrome which is characterised by loss of body weight, negative nitrogen balance and fatigue significantly affects patients' quality of life, morbidity and survival. Because the currently established therapeutical strategies are often disappointing many physicians tended to develop a therapeutical nihilism. Cancer anorexia and cachexia are two distinct syndromes which may have synergistic effects in a patient. This review highlights the growing understanding of the multidimensional pathophysiological background. An algorithm of the current treatment strategies is given. In addition, we discuss new anabolic and anticatabolic agents (e.g. eicosapentanoic acid) and the results from first clinical trials.

IT Major Concepts
 Gastroenterology (Human Medicine, Medical Sciences); Metabolism;
 Nutrition; Oncology (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms
 pancreas: digestive system, endocrine system

IT Diseases
anorexia: behavioral and mental disorders, nutritional
 disease
Anorexia (MeSH)

IT Diseases
 cachexia: nutritional disease
 Cachexia (MeSH)

IT Diseases
 pancreatic cancer: digestive system disease, endocrine
 disease/pancreas, neoplastic disease
 Pancreatic Neoplasms (MeSH)

IT Chemicals & Biochemicals
 eicosapentaenoic acid: metabolic-drug, clinical trial; ubiquitin

TI Tumour anorexia - tumour cachexia in case of gastrointestinal
 tumours: Standards and visions.
 Original Title: Tumoranorexie - Tumorkachexie bei gastrointestinalen
 Tumoren: Standards.

SO Zeitschrift fuer Gastroenterologie, (November 2002) Vol. 40, No.
 11, pp. 929-936. print.
 CODEN: ZGASAX. ISSN: 0044-2771.

AB. , . and survival. Because the currently established therapeutical
 strategies are often disappointing many physicians tended to develop a
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 distinct syndromes which may have synergistic effects in a patient. This
 review highlights the growing understanding. . . background. An
 algorithm of the current treatment strategies is given. In addition, we
 discuss new anabolic and anticatabolic agents (e.g.
eicosapentanoic acid) and the results from first
 clinical trials.

IT .
 Nutrition; Oncology (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms
 pancreas: digestive system, endocrine system

IT Diseases
anorexia: behavioral and mental disorders, nutritional
 disease
Anorexia (MeSH)

IT Diseases
 cachexia: nutritional disease
 Cachexia (MeSH)

IT Diseases
 pancreatic cancer: digestive system disease, endocrine
 disease/pancreas, neoplastic disease
 Pancreatic Neoplasms (MeSH)

L13 ANSWER 2 OF 11: BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation. on STN
 ACCESSION NUMBER: 2002:543209 BIOSIS
 DOCUMENT NUMBER: PREV200200543209
 TITLE: Cachexia related to mechanisms and nutrients.
 AUTHOR(S): Tisdale, M. J. [Reprint author]
 CORPORATE SOURCE: Pharmaceutical Sciences Research Institute, Aston
 University, Birmingham, UK
 m.j.tisdale@aston.ac.uk
 SOURCE: International Journal of Cancer Supplement, (2002
) No. 13, pp. 48. print..
 Meeting Info.: 18th UICC International Cancer Congress.

Oslo, Norway. June 30-July 05, 2002.

ISSN: 0898-6924.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Oct 2002
Last Updated on STN: 23 Oct 2002

SO International Journal of Cancer Supplement, (2002) No. 13, pp.
48. print.

Meeting Info.: 18th UICC International Cancer Congress. Oslo, Norway. June
30-July 05, 2002.

ISSN: 0898-6924.

IT Major Concepts

Metabolism; Nutrition; Oncology (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms

breast: reproductive system; lung: respiratory system; skeletal muscle:
muscular system; urine: excretory system; white adipose tissue

IT Diseases

anorexia: behavioral and mental disorders

Anorexia (MeSH)

IT Diseases

breast carcinoma: neoplastic disease, reproductive system
disease/female

Breast Neoplasms (MeSH); Carcinoma (MeSH)

IT Diseases

cancer cachexia: metabolic disease, neoplastic disease, nutritional
disease

Cachexia (MeSH)

IT Diseases

colon carcinoma: digestive system disease, neoplastic disease

Colonic Neoplasms (MeSH); Carcinoma (MeSH)

IT Diseases

liver carcinoma: digestive system disease, neoplastic disease

Carcinoma (MeSH); Liver Neoplasms (MeSH)

IT Diseases

lung carcinoma: neoplastic disease, respiratory system disease

Carcinoma (MeSH); Lung Neoplasms (MeSH)

IT Diseases

ovary carcinoma: neoplastic disease, reproductive system disease/female

IT Diseases

pancreas carcinoma: digestive system disease, neoplastic disease

Pancreatic Neoplasms (MeSH); Carcinoma (MeSH)

IT Chemicals & Biochemicals

15-hydroxyeicosatetranoic acid [15-HETE]: signaling molecule;

G-alpha-i: expression; G-alpha-s: expression; G-protein: expression;

eicosapentanoic acid: polyunsaturated fatty acid;

lipid metabolizing factor [LMF]; protein: catabolism;

proteolysis-inducing factor; triglyceride: hydrolysis

SO International Journal of Cancer Supplement, (2002) No. 13, pp.
48. print.

Meeting Info.: 18th UICC International Cancer Congress. Oslo, Norway. June
30-July 05, 2002.

ISSN: 0898-6924..

IT
of Organisms

breast: reproductive system; lung: respiratory system; skeletal muscle:
muscular system; urine: excretory system; white adipose tissue

IT Diseases

anorexia: behavioral and mental disorders

Anorexia (MeSH)

IT Diseases

breast carcinoma: neoplastic disease, reproductive system
disease/female
Breast Neoplasms (MeSH); Carcinoma (MeSH)

IT Diseases
cancer cachexia: metabolic disease,
disease
Pancreatic Neoplasms (MeSH); Carcinoma (MeSH)

IT Chemicals & Biochemicals
15-hydroxyeicosatetranoic acid [15-HETE]: signaling molecule;
G-alpha-i: expression; G-alpha-s: expression; G-protein: expression;
eicosapentanoic acid: polyunsaturated fatty acid;
lipid metabolizing factor [LMF]; protein: catabolism;
proteolysis-inducing factor; triglyceride: hydrolysis

L13 ANSWER 3 OF 11 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AB The progressive nutritional deterioration frequently found in cancer
patients, is often referred to as cancer cachexia. In contrast to
starvation, where it is possible to reverse the body composition changes
by the provision of extra calories, in cancer cachexia this reversal is
not observed, suggesting that anorexia alone is unlikely to be
responsible for this wasting syndrome. Over the past decades a number of
studies have focused on the possible mediators which may be responsible
for metabolic abnormalities observed in cancer patients. Pro-inflammatory
cytokines have been strongly implicated, but evidence supporting such a
direct role is lacking. Recently, exciting work regarding molecules
produced by tumour cells, and which may induce lipolysis and proteolysis,
has been published. There is also evidence that increased metabolism of
host resources may provide substrates which might promote tumour growth.
A number of studies have demonstrated that polyunsaturated fatty acids,
such as linoleic and arachidonic acid, are able to promote tumour cell
growth either by directly stimulating mitosis or by inhibiting apoptosis.
Even more interesting is the discovery of antagonists of these catabolic
factors such as eicosapentanoic acid for the lipolytic
factor, which may play a role in the treatment of these patients in the
near future.

ACCESSION NUMBER: 2001:35886 BIOSIS
DOCUMENT NUMBER: PREV200100035886
TITLE: Metabolic responses to tumour disease and progression:
Tumour-host interaction.
AUTHOR(S): Cravo, M. L. [Reprint author]; Gloria, L. M.; Claro, I.
CORPORATE SOURCE: Servico de Gastreenterologia, Instituto Portugues de
Oncologia Francisco Gentil, R. Professor Lima Basto, 1093,
Lisboa codex, Portugal
SOURCE: Clinical Nutrition (Edinburgh), (December, 2000)
Vol. 19, No. 6, pp. 459-465. print.
CODEN: CLNUDP. ISSN: 0261-5614.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Jan 2001
Last Updated on STN: 12 Feb 2002

SO Clinical Nutrition (Edinburgh), (December, 2000) Vol. 19, No. 6,
pp. 459-465. print.
CODEN: CLNUDP. ISSN: 0261-5614.

AB The progressive nutritional deterioration frequently found in cancer
patients, is often referred to as cancer cachexia. In contrast to
starvation, where it is possible to reverse the body composition changes
by the provision of extra calories, in cancer cachexia this reversal is
not observed, suggesting that anorexia alone is unlikely to be
responsible for this wasting syndrome. Over the past decades a number of
studies have focused on the possible mediators which may be responsible

for metabolic abnormalities observed in cancer patients. Pro-inflammatory cytokines have been strongly implicated, but evidence supporting such a direct role is lacking. Recently, exciting work regarding molecules produced by tumour cells, and which may induce lipolysis and proteolysis, has been published. There is also evidence that increased metabolism of host resources may provide substrates which might promote tumour growth. A number of studies have demonstrated that polyunsaturated fatty acids, such as linoleic and arachidonic acid, are able to promote tumour cell growth either by directly stimulating mitosis or by inhibiting apoptosis. Even more interesting is the discovery of antagonists of these catabolic factors such as eicosapentanoic acid for the lipolytic factor, which may play a role in the treatment of these patients in the near future.

SO Clinical Nutrition (Edinburgh), (December, 2000) Vol. 19, No. 6, pp. 459-465. print.

CODEN: CLNUDP. ISSN: 0261-5614.

AB. . . the body composition changes by the provision of extra calories, in cancer cachexia this reversal is not observed, suggesting that anorexia alone is unlikely to be responsible for this wasting syndrome. Over the past decades a number of studies have focused stimulating mitosis or by inhibiting apoptosis. Even more interesting is the discovery of antagonists of these catabolic factors such as eicosapentanoic acid for the lipolytic factor, which may play a role in the treatment of these patients in the near future.

L13 ANSWER 4 OF 11 USPATFULL on STN

AB A compound represented by the formula: ##STR1##

wherein R.sup.1 is an optionally substituted hydrocarbon group or an optionally substituted heterocyclic group; X is a bond, --CO--, --CH(OH)-- or a group represented by --NR.sup.6-- wherein R.sup.6 is a hydrogen atom or an optionally substituted alkyl group; n is an integer of 1 to 3; Y is an oxygen atom, a sulfur atom, --SO--, --SO.sub.2-- or a group represented by --NR.sup.7-- wherein R.sup.7 is a hydrogen atom or an optionally alkyl group; ring A is a benzene ring optionally having additional one to three substituents; p is an integer of 1 to 8; R.sup.2 is a hydrogen atom, an optionally substituted hydrocarbon group or an optionally substituted heterocyclic group; q is an integer of 0 to 6; m is 0 or 1; R.sup.3 is a hydroxy group, OR.sup.8 (R.sup.8 is an optionally substituted hydrocarbon group.) or NR.sup.9R.sup.10 (R.sup.9 and R.sup.10 are the same or different groups which are selected from a hydrogen atom, an optionally substituted hydrocarbon group, an optionally substituted heterocyclic group or an optionally substituted acyl group or R.sup.9 and R.sup.10 combine together to form a ring); R.sup.4 and R.sup.5 are the same or different groups which are selected from a hydrogen atom or an optionally substituted hydrocarbon group wherein R.sup.4 may form a ring with R.sup.2; provided that when R.sup.1 is a ethoxymethyl, a C.sub.1-3 alkyl, phenyl or p-methoxyphenyl and q=m=0, R.sup.3 is NR.sup.9R.sup.10; and provided that O-[2-chloro-4-(2-quinolylmethoxy)phenylmethyl]oxime and a methyl pyruvate of [2-chloro-4-(2-quinolylmethoxy)phenylmethyl]-2-iminoxypropionic acid are excluded; or a salt thereof which has excellent hypoglycemic and hypolipidemic actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:332756 USPATFULL

TITLE: Oxyiminoalkanoic acid derivatives

INVENTOR(S): Momose, Yu, Takarazuka, JAPAN

Odaka, Hiroyuki, Kobe, JAPAN

Imoto, Hiroshi, Kusatsu, JAPAN

Kimura, Hiroyuki, Sakai, JAPAN

PATENT ASSIGNEE(S): Sakamoto, Junichi, Toyonaka, JAPAN
Takeda Chemical Industries, Ltd., Osaka, JAPAN
(non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6495581	B1	20021217	<--
APPLICATION INFO.:	US 2000-714699		20001116	(9)
RELATED APPLN. INFO.:	Division of Ser. No. US 423854, now patented, Pat. No. US 6251926			

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1998-127921	19980511
	JP 1998-127922	19980511
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	McKane, Joseph K.	
ASSISTANT EXAMINER:	Wright, Sonya	
LEGAL REPRESENTATIVE:	Chao, Mark, Ramesh, Elaine M.	
NUMBER OF CLAIMS:	22	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	0 Drawing Figure(s); 0 Drawing Page(s)	
LINE COUNT:	5850	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 6495581 B1 20021217 <--

SUMM A compound according to the invention may also be employed as a pharmaceutical for controlling appetite, food intake, diet and anorexia.

SUMM In addition, an agent whose cachexia improving effect has been established in an animal model or at a clinical stage, such as a cyclooxygenase inhibitor (e.g., indomethacin and the like) [Cancer Research, Vol.49, page 5935-5939, 1989], a progesterone derivative (e.g., megestrol acetate) [Journal of Clinical Oncology, Vol.12, page 213-225, 1994], a glucosteroid (e.g., dexamethasone and the like), a metoclopramide-based agent, a tetrahydrocannabinol-based agent (supra), a lipid metabolism improving agent (e.g., eicosapentanoic acid and the like) [British Journal of Cancer), Vol.68, page 314-318, 1993], a growth hormone, IGF-1, or an antibody against TNF- α , LIF, IL-6, oncostatin M which are cachexia-inducing factors may also be employed concomitantly with a compound according to the present invention.

DETD A compound according to the invention may also be employed as a pharmaceutical for controlling appetite or food intake, diet and anorexia.

PI US 6495581 B1 20021217 <--

SUMM A compound according to the invention may also be employed as a pharmaceutical for controlling appetite, food intake, diet and anorexia.

SUMM . . . a glucosteroid (e.g., dexamethasone and the like), a metoclopramide-based agent, a tetrahydrocannabinol-based agent (supra), a lipid metabolism improving agent (e.g., eicosapentanoic acid and the like) [British Journal of Cancer), Vol.68, page 314-318, 1993], a growth hormone, IGF-1, or an antibody against TNF- α ,

DETD . . . compound according to the invention may also be employed as a pharmaceutical for controlling appetite or food intake, diet and anorexia.

L13 ANSWER 5 OF 11 USPATFULL on STN

AB A medicinal composition for the prophylaxis and treatment of cachexia which comprises a compound of the formula: ##STR1##

wherein R represents a hydrocarbon group that may be substituted or a heterocyclic group that may be substituted; Y represents a group of the formula --CO--, --CH(OH)--, or --NR^{sup.3}-- (R^{sup.3} represents an alkyl group that may be substituted); m is 0 or 1; n is 0, 1 or 2; X represents CH or N; A represents a bond or a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms; Q represents oxygen or sulfur; R^{sup.1} represents hydrogen or an alkyl group; ring E may have further 1 to 4 substituents, which may form a ring in combination with R^{sup.1}; L and M respectively represent hydrogen or may be combined with each other to form a bond, provided that when m and n are 0, X represents CH; A represents a bond, Q represents sulfur, R^{sup.1}, L and M respectively represent hydrogen, and ring E does not have further substituents, R does not represent dihydrobenzopyranyl; or a salt thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:70000 USPATFULL

TITLE: Anticachectic composition

INVENTOR(S): Momose, Yu, Takarazuka, JAPAN
Matsutani, Etsuya, Suita, JAPAN
Sohda, Takashi, Takatsuki, JAPAN

PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, JAPAN
(non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6365607	B1	20020402	<--
APPLICATION INFO.:	US 2000-605628		20000628	(9)
RELATED APPLN. INFO.:	Division of Ser. No. US 155593, now patented, Pat. No. US 6110948			

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1996-82845	19960404
	JP 1997-27957	19970212
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Goldberg, Jerome D.	
LEGAL REPRESENTATIVE:	Wenderoth, Lind & Ponack, L.L.P.	
NUMBER OF CLAIMS:	16	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	0 Drawing Figure(s); 0 Drawing Page(s)	
LINE COUNT:	829	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 6365607 B1 20020402 <--

SUMM Cachexia is a systemic syndrome with progressive loss of body weight, anemia, edema, and anorexia as cardinal symptoms which develops in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome [e.g. Kern et al., Cancer Cachexia, J. Parenteral and Enteral Nutrition, 12, 286-298 (1988) and American Journal of Medicine, 85, 289-291 (1988)].

SUMM Compound (I) or a salt thereof of the present invention (hereinafter referred to as compound of the present invention) have anticachectic activity, that is the activity to relieve the systemic syndrome featuring progressive loss of body weight (inclusive of weight loss due

to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome. In addition, the toxic potential of the compound of the present invention is low.

SUMM Furthermore, drugs which are documented as being anticachectic in an animal model or clinically, such as cyclooxygenase inhibitors (e.g. indomethacin) [Cancer Research, 49, 5935-5939, (1989)], progesterone derivatives (e.g. megestrol acetate) [Journal of Clinical Oncology, 12, 213-225, 1994], glucocorticoids (e.g. dexamethasone), metoclopramides, tetrahydrocannabinols (the same literature as above), lipid metabolism improving agents (e.g. eicosapentanoic acid) [British Journal of Cancer, 68, 314-318, 1993], growth hormone, IGF-1, and antibodies to the cachexia-inducing factors TNF- α , LIF, IL-6, and oncostatin M may also be used together with the composition for prophylaxis and treatment of the present invention.

SUMM Among the compound of the present invention, especially a compound which has a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms for A in the formula (I) or a salt thereof, has an activity to prevent and treat atherosclerosis, and an activity to regulate appetite and food intake in disorders associated with under-eating, such as anorexia nervosa, and disorders associated with over-eating, such as obesity and anorexia bulimia. Therefore, such a compound or a salt thereof can be used, as such or by providing in a pharmaceutical dosage form in the same manner as described above, as an agent for prophylaxis and treatment of atherosclerosis, or medicine for the regulation of appetite and food intake.

DETD The composition for prophylaxis and treatment of the present invention is of value as an agent for prophylaxis and treatment of cachexia which develops in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome. The composition for prophylaxis and treatment of the present invention is conducive to relief of the systemic syndrome, the cardinal signs of which are progressive loss of body weight (inclusive of weight loss due to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia, in said chronic diseases.

CLM What is claimed is:

4. A method for treating anorexia in a diabetic mammal, which comprises administering to said mammal an effective amount of a compound of the formula: ##STR11## wherein R represents a hydrocarbon group that may be substituted or a heterocyclic group that may be substituted; Y represents a group of the formula --CO--, --CH(OH)--, or --NR³-- wherein R³ represents an alkyl group that may be substituted; m is 0 or 1; n is 0, 1 or 2; X represents CH or N; A represents a bond or a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms; Q represents oxygen or sulfur; R¹ represents hydrogen or an alkyl group; ring E may have further 1 to 4 substituents, which may form a ring in combination with R¹; L and M respectively represent hydrogen or may be combined with each other to form a bond; provided that R does not represent dihydrobenzopyranyl when m and n are 0, X represents CH, A represents a bond, Q represents sulfur, R¹, L and M respectively represent hydrogen, and ring E does not have further substituents; or a salt thereof.

PI US 6365607 B1 20020402 <--|

SUMM Cachexia is a systemic syndrome with progressive loss of body weight, anemia, edema, and anorexia as cardinal symptoms which

develops in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, . . .

SUMM . . . loss of body weight (inclusive of weight loss due to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome. In . . .

SUMM . . . Clinical Oncology, 12, 213-225, 1994], glucocorticoids (e.g. dexamethasone), metoclopramides, tetrahydrocannabinols (the same literature as above), lipid metabolism improving agents (e.g. eicosapentanoic acid) [British Journal of Cancer, 68, 314-318, 1993], growth hormone, IGF-1, and antibodies to the cachexia-inducing factors TNF- α , LIF, IL-6, and . . .

SUMM . . . prevent and treat atherosclerosis, and an activity to regulate appetite and food intake in disorders associated with under-eating, such as anorexia nervosa, and disorders associated with over-eating, such as obesity and anorexia bulimia. Therefore, such a compound or a salt thereof can be used, as such or by providing in a pharmaceutical dosage. . .

DETD . . . loss of body weight (inclusive of weight loss due to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia, in said chronic diseases.

4. A method for treating anorexia in a diabetic mammal, which comprises administering to said mammal an effective amount of a compound of the formula: ##STR11##.

L13 ANSWER 6 OF 11 USPATFULL on STN

AB Method of treating or ameliorating symptoms of T-cell mediated disease wherein a composition comprising a therapeutically effective amount of a polyunsaturated fatty acid and a pharmaceutically acceptable carrier is administered to the patient. The polyunsaturated fatty acid contains 18-25 carbon atoms, 1-6 double bonds and has 1 or 2 substitutions selected from β oxa, γ oxa, β thia and γ thia, based on the fatty acid acyl carbon atom, or the polyunsaturated fatty acid contains 16-26 carbon atoms, 3-double bonds and is covalently coupled at the carboxylic acid group to an amino acid.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2001:112384 USPATFULL

TITLE: Methods of treating immunopathologies using polyunsaturated fatty acids

INVENTOR(S): Ferrante, Antonio, Mount Osmond, Australia
Poulos, Alfred, Kensington Gardens, Australia
Pitt, Michael Joseph, Gordon, Australia
Easton, Christopher John, Weetangera, Australia
Sleigh, Marilyn Joy, Neutral Bay, Australia
Rathjen, Deborah Ann, Sheidow Park, Australia
Widmer, Fred, Ryde, Australia

PATENT ASSIGNEE(S): Peptide Technology Limited, New South Wales, Australia (non-U.S. corporation)
Women's and Children's Hospital Adelaide, North Adelaide, Australia (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6262119	B1	20010717	<--
	WO 9738688		19971023	<--
APPLICATION INFO.:	US 1999-171095		19990412	(9)
	WO 1997-AU231		19970414	
			19990412	PCT 371 date
			19990412	PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	AU 1996-9250	19960412
	AU 1996-9538	19960426
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Krass, Frederick	
LEGAL REPRESENTATIVE:	Nixon & Vanderhye	
NUMBER OF CLAIMS:	13	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	46 Drawing Figure(s); 11 Drawing Page(s)	
LINE COUNT:	1532	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI	US 6262119	B1	20010717	<--
	WO 9738688	19971023		<--

SUMM Rheumatoid arthritis (Marrow et al, I "Auto-immune Rheumatic Disease", Blackwell Scientific Publ. Oxford, UK, Chapter 4 ppl48-207 (1987) is a disease characterised by chronic inflammation and erosion of joints that may affect up to 3% of the population, including children. Symptoms of rheumatoid arthritis include morning stiffness, swelling and pain upon motion in at lease one joint and joint swelling. Non-specific symptoms including lethargy, anorexia and weakness as well as fever and lymphadenopathy (characteristic of immune activation) may antedate joint involvement. Extra-articular manifestations of rheumatoid arthritis include vasculitis, cataracts, uveitis, interstitial fibrosis, pericarditis and myocarditis, peripheral neuropathy, myeloid deposits, chronic anaemia and subcutaneous and pulmonary nodules.

SUMM Cachexia, which is characteristic of chronic exposure to TNF or interleukin-6, is a common symptom of advanced malignancy and severe infection. It is characterised by abnormal protein and glucose metabolism and body wasting. Chronic administration of TNF IL-1 in mice, rats and/or humans cause anorexia, weight loss and depletion of body lipid and protein within 7 to 10 days (Cerami et al, 1985, Immunol. Lett, 11, 173; Fong et al, 1989 J. Exp. Med. 170, 1627. Moldawer et al, Am. J. Physiol., 254 G450-G456, 1988; Fong et al, Am. J. Physiol. 256, R659-R665 (1989); McCarthy et al, Am. J. Clin. Nature. 42, 1179-1182. 1982). TNF levels have been measured in patients with cancer and chronic disease associated with cachexia.

DETD TABLE 6

EFFECT OF AMINO ACID CONJUGATED PUFAS ON

PHA-STIMULATED TNF α AND INTERFERON γ PRODUCTION

COMPOUND	% inhibition of	
	TNF α production	IFN γ production
α -linolenic acid-glycine	29.3	14.5
α -linolenic acid-aspartic acid	0	0
γ -linolenic acid-glycine	21.5	0
γ -linolenic acid-aspartic acid	4.7	0
arachidonic acid-glycine	26.6	35.9
arachidonic acid-aspartic acid	38.3	68.4
<u>eicosapentanoic acid</u> -glycine	11	68.2
<u>eicosapentanoic acid</u> -aspartic acid	17.1	66.1
docosahexanoic acid-glycine	16.2	44
docosahexanoic acid-aspartic acid	17.4	8.3

All PUFA were at 20 μ M

DETD TABLE 7

EFFECT OF PUFA ON CELL PROLIFERATION INDUCED BY PHA

COMPOUND	% INHIBITION OF PROLIFERATION	
α-linolenic acid-glycine	15.6	
α-linolenic acid-aspartic acid	7.3	
γ-linolenic acid-glycine	29	
γ-linolenic acid-aspartic acid	15.4	
arachidonic acid-glycine	8	
arachidonic acid-aspartic acid	39/7	
<u>eicosapentanoic acid</u> -glycine	5.4	
<u>eicosapentanoic acid</u> -aspartic acid	20.7	
docosahexanoic acid-glycine	16.6	
docosahexanoic acid-aspartic acid	21.1	

All PUFA were at 20 μM

PI US 6262119 B1 20010717 <--
WO 9738688 19971023 <--

SUMM . . . include morning stiffness, swelling and pain upon motion in at least one joint and joint swelling. Non-specific symptoms including lethargy, anorexia and weakness as well as fever and lymphadenopathy (characteristic of immune activation) may antedate joint involvement. Extra-articular manifestations of rheumatoid.

SUMM . . . by abnormal protein and glucose metabolism and body wasting. Chronic administration of TNF IL-1 in mice, rats and/or humans cause anorexia, weight loss and depletion of body lipid and protein within 7 to 10 days (Cerami et al, 1985, Immunol. Lett., . . .

DETD . . . acid-glycine	29.3	14.5	
α-linolenic acid-aspartic acid	0	0	
γ-linolenic acid-glycine	21.5	0	
γ-linolenic acid-aspartic acid	4.7	0	
arachidonic acid-glycine	26.6	35.9	
arachidonic acid-aspartic acid	38.3	68.4	
<u>eicosapentanoic acid</u> -glycine	11	68.2	
<u>eicosapentanoic acid</u> -aspartic acid	17.1		66.1
docosahexanoic acid-glycine	16.2	44	
docosahexanoic acid-aspartic acid	17.4		8.3

All PUFA were at 20 μM

DETD . . . acid-glycine 15.6
α-linolenic acid-aspartic acid 7.3
γ-linolenic acid-glycine 29
γ-linolenic acid-aspartic acid 15.4
arachidonic acid-glycine 8
arachidonic acid-aspartic acid 39/7
eicosapentanoic acid-glycine 5.4
eicosapentanoic acid-aspartic acid 20.7
docosahexanoic acid-glycine 16.6
docosahexanoic acid-aspartic acid 21.1

All PUFA were at 20 μM

L13 ANSWER 7 OF 11 USPATFULL on STN

AB This invention provides a novel oxyiminoalkanoic acid derivative which has excellent hypoglycemic and hypolipidemic actions and which is used for the treatment of diabetes mellitus, hyperlipemia, insulin insensitivity, insulin resistance and impaired glucose tolerance.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2001:97948 USPATFULL

TITLE: Oxyiminoalkanoic acid derivatives with hypoglycemic and hypolipidemic activity

INVENTOR(S): Momose, Yu, Takarazuka, Japan
Odaka, Hiroyuki, Kobe, Japan
Imoto, Hiroshi, Kusatsu, Japan
Kimura, Hiroyuki, Sakai, Japan

PATENT ASSIGNEE(S): Sakamoto, Junichi, Toyonaka, Japan
Takeda Chemical Industries, Ltd., Osaka, Japan
(non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6251926	B1	20010626	<--
	WO 9958510		19991118	<--
APPLICATION INFO.:	US 1999-423854		19991115	(9)
	WO 1999-JP2407		19990510	
			19991115	PCT 371 date
			19991115	PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1998-127921	19980511
	JP 1998-127922	19980511

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Powers, Fiona T.
ASSISTANT EXAMINER: Wright, Sonya
LEGAL REPRESENTATIVE: Riesen, Philippe Y.
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
LINE COUNT: 5841

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 6251926 B1 20010626 <--
WO 9958510 19991118 <--

SUMM A compound according to the invention may also be employed as a pharmaceutical for controlling appetite, food intake, diet and anorexia.

SUMM In addition, an agent whose cachexia improving effect has been established in an animal model or at a clinical stage, such as a cyclooxygenase inhibitor (e.g., indomethacin and the like) [Cancer Research, Vol.49, page 5935-5939, 1989], a progesterone derivative (e.g., megestrol acetate) [Journal of Clinical Oncology, Vol.12, page 213-225, 1994], a glucosteroid (e.g., dexamethasone and the like), a metoclopramide-based agent, a tetrahydrocannabinol-based agent (supra), a lipid metabolism improving agent (e.g., eicosapentanoic acid and the like) [British Journal of Cancer), Vol.68, page 314-318, 1993], a growth hormone, IGF-1, or an antibody against TNF- α , LIF, IL-6, oncostatin M which are cachexia-inducing factors may also be employed concomitantly with a compound according to the present invention.

DETD A compound according to the invention may also be employed as a pharmaceutical for controlling appetite or food intake, diet and anorexia.

PI US 6251926 B1 20010626 <--
WO 9958510 19991118 <--

SUMM A compound according to the invention may also be employed as a pharmaceutical for controlling appetite, food intake, diet and anorexia.

SUMM . . . a glucosteroid (e.g., dexamethasone and the like), a metoclopramide-based agent, a tetrahydrocannabinol-based agent (supra), a lipid metabolism improving agent (e.g., eicosapentanoic acid and the like) [British Journal of Cancer), Vol.68, page 314-318, 1993], a growth hormone, IGF-1, or an antibody against TNF- α ,

DETD . . . compound according to the invention may also be employed as a

pharmaceutical for controlling appetite or food intake, diet and
anorexia.

L13 ANSWER 8 OF 11 USPATFULL on STN

AB A medicinal composition for the prophylaxis and treatment of cachexia which comprises a compound of the formula: ##STR1## wherein R represents a hydrocarbon group that may be substituted or a heterocyclic group that may be substituted; Y represents a group of the formula --CO--, --CH(OH)--, or --NR^{sup.3}-- (R^{sup.3} represents an alkyl group that may be substituted); m is 0 or 1; n is 0, 1 or 2; X represents CH or N; A represents a bond or a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms; Q represents oxygen or sulfur; R^{sup.1} represents hydrogen or an alkyl group; ring E may have further 1 to 4 substituents, which may form a ring in combination with R^{sup.1}; L and M respectively represent hydrogen or may be combined with each other to form a bond, provided that when m and n are 0, X represents CH, A represents a bond, Q represents sulfur, R^{sup.1}, L and M respectively represent hydrogen, and ring E does not have further substituents, R does not represent dihydrobenzopyranyl; or a salt thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2000:113975 USPATFULL
TITLE: Anticachectic composition
INVENTOR(S): Momose, Yu, Takarazuka, Japan
Matsutani, Etsuya, Suita, Japan
Sohda, Takashi, Takatsuki, Japan
PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, Japan
(non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6110948		20000829	<--
	WO 9737656		19971016	<--
APPLICATION INFO.:	US 1998-155593		19980930	(9)
	WO 1997-JP1148		19970403	
			19980930	PCT 371 date
			19980930	PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1996-82845	19960404
	JP 1997-27957	19970212
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Goldberg, Jerome D.	
LEGAL REPRESENTATIVE:	Wenderoth, Lind & Ponack, LLP.	
NUMBER OF CLAIMS:	11	
EXEMPLARY CLAIM:	1	
LINE COUNT:	779	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 6110948 20000829 <--
WO 9737656 19971016 <--

SUMM Cachexia is a systemic syndrome with progressive loss of body weight, anemia, edema, and anorexia as cardinal symptoms which develops in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome [e.g. Kern et al., Cancer Cachexia, J. Parenteral and Enteral Nutrition, 12, 286-298 (1988) and American Journal of Medicine, 85, 289-291 (1988)].

SUMM Compound (I) or a salt thereof of the present invention (hereinafter

referred to as compound of the present invention) have anticachectic activity, that is the activity to relieve the systemic syndrome featuring progressive loss of body weight (inclusive of weight loss due to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome. In addition, the toxic potential of the compound of the present invention is low.

SUMM Furthermore, drugs which are documented as being anticachectic in an animal model or clinically, such as cyclooxygenase inhibitors (e.g. indomethacin) [Cancer Research, 49, 5935-5939, (1989)], progesterone derivatives (e.g. megestrol acetate) [Journal of Clinical Oncology, 12, 213-225, 1994], glucocorticoids (e.g. dexamethasone), metoclopramides, tetrahydrocannabinols (the same literature as above), lipid metabolism improving agents (e.g. eicosapentanoic acid) [British Journal of Cancer, 68, 314-318, 1993], growth hormone, IGF-1, and antibodies to the cachexia-inducing factors TNF- α , LIF, IL-6, and oncostatin M may also be used together with the composition for prophylaxis and treatment of the present invention.

SUMM Among the compound of the present invention, especially a compound which has a bivalent aliphatic hydrocarbon group having 1 to 7 carbon atoms for A in the formula (I) or a salt thereof, has an activity to prevent and treat atherosclerosis, and an activity to regulate appetite and food intake in disorders associated with under-eating, such as anorexia nervosa, and disorders associated with over-eating, such as obesity and anorexia bulimia. Therefore, such a compound or a salt thereof can be used, as such or by providing in a pharmaceutic dosage form in the same manner as described above, as an agent for prophylaxis and treatment of atherosclerosis, or medicine for the regulation of appetite and food intake.

DETD The composition for prophylaxis and treatment of the present invention is of value as an agent for prophylaxis and treatment of cachexia which develops in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome. The composition for prophylaxis and treatment of the present invention is conducive to relief of the systemic syndrome, the cardinal signs of which are progressive loss of body weight (inclusive of weight loss due to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia, in said chronic diseases.

PI US 6110948 20000829 <--
WO 9737656 19971016 <--

SUMM Cachexia is a systemic syndrome with progressive loss of body weight, anemia, edema, and anorexia as cardinal symptoms which develops in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease,

SUMM . . . loss of body weight (inclusive of weight loss due to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia in chronic diseases such as malignant tumor, tuberculosis, diabetes, blood dyscrasia, endocrine disease, infectious disease, and acquired immunodeficiency syndrome. In

SUMM . . . Clinical Oncology, 12, 213-225, 1994], glucocorticoids (e.g. dexamethasone), metoclopramides, tetrahydrocannabinols (the same literature as above), lipid metabolism improving agents (e.g. eicosapentanoic acid) [British Journal of Cancer, 68, 314-318, 1993], growth hormone, IGF-1, and antibodies to the cachexia-inducing factors TNF- α , LIF, IL-6, and

SUMM . . . prevent and treat atherosclerosis, and an activity to regulate

appetite and food intake in disorders associated with under-eating, such as anorexia nervosa, and disorders associated with over-eating, such as obesity and anorexia bulimia. Therefore, such a compound or a salt thereof can be used, as such or by providing in a pharmaceutic dosage.

DETD loss of body weight (inclusive of weight loss due to lipolysis and weight loss due to myolysis), anemia, edema, and anorexia, in said chronic diseases.

L13 ANSWER 9 OF 11 USPATFULL on STM

AB Enteral nutritional support for a person infected with human immunodeficiency virus is provided by including in the diet a nutritional product which contains a soy protein hydrolysate having a degree of hydrolysis in the range of about 14 to 17 and a molecular weight partition, as determined by size exclusion chromatography, wherein 30-60% of the particles have a molecular weight in the range of 1500-5000 Daltons. The nutritional product also contains a source of intact protein. The nutritional product has a ratio, by weight, of n-6 to n-3 fatty acids of about 1.3:1 to 2.5:1. The nutritional product also contains a source of dietary fiber.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 96:1451 USPATFULL

TITLE: Method of providing enteral nutritional support to persons infected with human immunodeficiency virus
INVENTOR(S): Cope, Frederick O., Worthington, OH, United States
DeWille, Normanella T., Upper Arlington, OH, United States

Richards, Ernest W., Columbus, OH, United States
Mazer, Terrence B., Reynoldsburg, OH, United States
Abbruzzese, Bonnie C., Dublin, OH, United States
Snowden, Gregory A., Pickerington, OH, United States
Chandler, Michael A., Gahanna, OH, United States

PATENT ASSIGNEE(S): Abbott Laboratories, Abbott Park, IL, United States
(U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5480872		19960102	<--
APPLICATION INFO.:	US 1993-69066		19930528	(8)
DISCLAIMER DATE:	20110719			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Sayala, Chhaya D.			
LEGAL REPRESENTATIVE:	Drayer, Lonnie R.			
NUMBER OF CLAIMS:	20			
EXEMPLARY CLAIM:	1			
LINE COUNT:	1369			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5480872 19960102 <--

SUMM Trujillo et al, "Assessment of nutritional status, nutrient intake, and nutrition support in AIDS patients", JOURNAL OF THE AMERICAN DIETETIC ASSOCIATION, Vol.92, No.4, pages 477-478 (1992) reports observations that hospitalized AIDS patients can consume only 70% of estimated basal energy needs and 65% of protein needs, which does not account for the increased needs of hypermetabolism associated with acute infection or any physical activity. Patients with AIDS have moderate to severe metabolic stress similar to that found in other critically ill patients. This stress, coupled with the anorexia and malabsorption associated with the disease, promotes malnutrition. Irrespective of any possible specific relationship between nutrition and the HIV disease

process, malnourished patients will be debilitated and unable to function optimally. Malnutrition in general affects five areas of functionality: reproductive competence, immunocompetence, work performance and/or behavioral performance and cognition. THE FASEB JOURNAL, Vol. 5, No. 10, pages 2329-2330, at page 2330 (1991)

DETD The refined deodorized fish oil used in the nutritional product of the present invention contains about 45% total n-3 polyunsaturated fatty acids, of which about 28% are Eicosapentanoic acid (EPA, C20:5n-3) and 13% are Docosahexanoic acid (DHA, C22:6n-3). These unsaturated fatty acids make the oil very prone to oxidation. Thus, approximately 7000 ppm of natural mixed tocopherols are added to the oil by the supplier before shipment to prevent oxidation. Upon arrival the oil is kept under nitrogen and refrigeration until it is used. The shelf-life assigned to this commodity is only 70 days to assure that the oil is of optimal quality at the time of use. The nutritional product has been manufactured using a fish oil manufactured from sardines and has been obtained from Mochida International in Shijuku-Ku, Tokyo, Japan.

DETD The concentrated levels of vitamins and minerals, which are presented in TABLE 1, enable the nutritional product of the present invention to meet the nutritional requirements of enterally fed patients with a smaller volume of the product. HIV infected patients often need volume restriction because of gastrointestinal tract problems, or drug-or treatment-related anorexia, nausea, and/or vomiting.

PI US 5480872 19960102 <--

SUMM . . . have moderate to severe metabolic stress similar to that found in other critically ill patients. This stress, coupled with the anorexia and malabsorption associated with the disease, promotes malnutrition. Irrespective of any possible specific relationship between nutrition and the HIV disease.

DETD . . . the nutritional product of the present invention contains about 45% total n-3 polyunsaturated fatty acids, of which about 28% are Eicosapentanoic acid (EPA, C20:5n-3) and 13% are Docosahexanoic acid (DHA, C22:6n-3). These unsaturated fatty acids make the oil very prone to oxidation.

DETD . . . smaller volume of the product. HIV infected patients often need volume restriction because of gastrointestinal tract problems, or drug-or treatment-related anorexia, nausea, and/or vomiting.

L13 ANSWER 10 OF 11 USPATFULL on STN

AB An enteral nutritional product for persons infected with human immunodeficiency virus contains a soy protein hydrolysate having a degree of hydrolysis in the range of about 14 to 17 and a molecular weight partition, as determined by size exclusion chromatography, wherein 30-60% of the particles have a molecular weight in the range of 1500-5000 Daltons. The nutritional product also contains a source of intact protein. The nutritional product has a ratio, by weight, of n-6 to n-3 fatty acids of about 1.3:1 to 2.5:1. The nutritional product also contains a source of dietary fiber.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 95:29628 USPATFULL

TITLE: Nutritional product for persons infected with human immunodeficiency virus

INVENTOR(S): Cope, Frederick O., Worthington, OH, United States
DeWille, Normanella T., Upper Arlington, OH, United States
Richards, Ernest W., Columbus, OH, United States
Mazer, Terrence B., Reynoldsburg, OH, United States
Abbruzzese, Bonnie C., Dublin, OH, United States

PATENT ASSIGNEE(S): Snowden, Gregory A., Pickerington, OH, United States
 Chandler, Michael A., Gahanna, OH, United States
 Abbott Laboratories, Abbott Park, IL, United States
 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5403826		19950404 <--
APPLICATION INFO.:	US 1993-69269		19930528 (8)
DISCLAIMER DATE:	20110719		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Wityshyn, Michael G.		
ASSISTANT EXAMINER:	Sayala, C.		
LEGAL REPRESENTATIVE:	Drayer, Lonnie R., Nickey, Donald O.		
NUMBER OF CLAIMS:	20		
EXEMPLARY CLAIM:	1		
LINE COUNT:	1375		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5403826 19950404 <--

SUMM Trujillo et al, "Assessment of nutritional status, nutrient intake, and nutrition support in AIDS patients", JOURNAL OF THE AMERICAN DIETETIC ASSOCIATION, Vol.92, No.4, pages 477-478 (1992) reports observations that hospitalized AIDS patients can consume only 70% of estimated basal energy needs and 65% of protein needs, which does not account for the increased needs of hypermetabolism associated with acute infection or any physical activity. Patients with AIDS have moderate to severe metabolic stress similar to that found in other critically ill patients. This stress, coupled with the anorexia and malabsorption associated with the disease, promotes malnutrition. Irrespective of any possible specific relationship between nutrition and the HIV disease process, malnourished patients will be debilitated and unable to function optimally. Malnutrition in general affects five areas of functionality: reproductive competence, immunocompetence, work performance and/or behavioral performance and cognition. THE FASEB JOURNAL, Vol. 5, No. 10, pages 2329-2330, at page 2330 (1991)

DETD The refined deodorized fish oil used in the nutritional product of the present invention contains about 45% total n-3 polyunsaturated fatty acids, of which about 28% are Eicosapentanoic acid (EPA, C20:5n-3) and 13% are Docosahexanoic acid (DHA, C22:6n-3). These unsaturated fatty acids make the oil very prone to oxidation. Thus, approximately 7000 ppm of natural mixed tocopherols are added to the oil by the supplier before shipment to prevent oxidation. Upon arrival the oil is kept under nitrogen and refrigeration until it is used. The shelf-life assigned to this commodity is only 70 days to assure that the oil is of optimal quality at the time of use. The nutritional product has been manufactured using a fish oil manufactured from sardines and has been obtained from Mochida International in Shijuku-Ku, Tokyo, Japan.

DETD The concentrated levels of vitamins and minerals, which are presented in TABLE 1, enable the nutritional product of the present invention to meet the nutritional requirements of enterally fed patients with a smaller volume of the product. HIV infected patients often need volume restriction because of gastrointestinal tract problems, or drug-or treatment-related anorexia, nausea, and/or vomiting.

PI US 5403826 19950404 <--

SUMM have moderate to severe metabolic stress similar to that found in other critically ill patients. This stress, coupled with the anorexia and malabsorption associated with the disease, promotes malnutrition. Irrespective of any possible specific relationship between

nutrition and the HIV disease.

DETD . . . the nutritional product of the present invention contains about 45% total n-3 polyunsaturated fatty acids, of which about 28% are Eicosapentanoic acid (EPA, C20:5n-3) and 13% are Docosahexanoic acid (DHA, C22:6n-3). These unsaturated fatty acids make the oil very prone to oxidation.

DETD . . . smaller volume of the product. HIV infected patients often need volume restriction because of gastrointestinal tract problems, or drug-or treatment-related anorexia, nausea, and/or vomiting.

L13 ANSWER 11 OF 11 USPATFULL on STN

AB The apoptosis of CD4 cells in a person infected with the human immunodeficiency virus may be impeded by enterally feeding to the infected person a nutritional product which contains soy protein hydrolysate having a degree of hydrolysis in the range of about 14 to 17 and a molecular weight partition, as determined by size exclusion chromatography, wherein 30%-60% of the particles have a molecular weight in the range of 1500-5000 Daltons. The nutritional product also contains a source of intact protein. The nutritional product has a ratio, by weight, of n-6 to n-3 fatty acids of about 1.3:1 to 2.5:1. The nutritional product also contains a source of dietary fiber.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 94:62434 USPATFULL

TITLE: Method of impeding apoptosis of CD4 cells in persons infected with human immunodeficiency virus

INVENTOR(S): Cope, Frederick O., Worthington, OH, United States

PATENT ASSIGNEE(S): Abbott Laboratories, Abbott Park, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5330972		19940719	<--
APPLICATION INFO.:	US 1993-69264		19930528	(8)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Schain, Howard E.			
ASSISTANT EXAMINER:	Koh, Choon Park			
LEGAL REPRESENTATIVE:	Drayer, Lonnie R., Nickey, Donald O.			
NUMBER OF CLAIMS:	16			
EXEMPLARY CLAIM:	1			
LINE COUNT:	1305			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5330972 19940719 <--

SUMM Trujillo et al, "Assessment of nutritional status, nutrient intake, and nutrition support in AIDS patients", JOURNAL OF THE AMERICAN DIETETIC ASSOCIATION, Vol.92, No.4, pages 477-478 (1992) reports observations that hospitalized AIDS patients can consume only 10% of estimated basal energy needs and 65% of protein needs, which does not account for the increased needs of hypermetabolism associated with acute infection or any physical activity. Patients with AIDS have moderate to severe metabolic stress similar to that found in other critically ill patients. This stress, coupled with the anorexia and malabsorption associated with the disease, promotes malnutrition. Irrespective of any possible specific relationship between nutrition and the HIV disease process, malnourished patients will be debilitated and unable to function optimally. Malnutrition in general affects five areas of functionality: reproductive competence, immunocompetence, work performance and/or behavioral performance and cognition. THE FASEB JOURNAL, Vol. 5, No. 10, pages 2329-2330, at page 2330 (1991)

DETD The refined deodorized fish oil used in the nutritional product of the present invention contains about 45% total n-3 polyunsaturated fatty acids, of which about 28% are Eicosapentanoic acid (EPA, C20:5n-3) and 13% are Docosahexanoic acid (DHA, C22:6n-3). These unsaturated fatty acids make the oil very prone to oxidation. Thus, approximately 7000 ppm of natural mixed tocopherols are added to the oil by the supplier before shipment to prevent oxidation. Upon arrival the oil is kept under nitrogen and refrigeration until it is used. The shelf-life assigned to this commodity is only 10 days to assure that the oil is of optimal quality at the time of use. The nutritional product has been manufactured using a fish oil manufactured from sardines and has been obtained from Mochida International in Shijuku-Ku, Tokyo, Japan.

DETD The concentrated levels of vitamins and minerals, which are presented in TABLE 1, enable the nutritional product of the present invention to meet the nutritional requirements of enterally fed patients with a smaller volume of the product. HIV infected patients often need volume restriction because of gastrointestinal tract problems, or drug-or treatment-related anorexia, nausea, and/or vomiting.

PI US 5330972 19940719 <--

SUMM . . . have moderate to severe metabolic stress similar to that found in other critically ill patients. This stress, coupled with the anorexia and malabsorption associated with the disease, promotes malnutrition. Irrespective of any possible specific relationship between nutrition and the HIV disease. . . .

DETD . . . the nutritional product of the present invention contains about 45% total n-3 polyunsaturated fatty acids, of which about 28% are Eicosapentanoic acid (EPA, C20:5n-3) and 13% are Docosahexanoic acid (DHA, C22:6n-3). These unsaturated fatty acids make the oil very prone to oxidation. Thus, approximately. . . .

DETD . . . a smaller volume of the product. HIV infected patients often need volume restriction because of gastrointestinal tract problems, or drug-or treatment-related anorexia, nausea, and/or vomiting.

=> fil reg

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

88.16

105.67

FILE 'REGISTRY' ENTERED AT 13:15:56 ON 24 AUG 2007

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DICTIONARY FILE UPDATES: 23 AUG 2007 HIGHEST RN 945525-31-5

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REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information

on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stngen/stndoc/properties.html>

=> e Eicosapentaenoic acid/cn

E1 1 EICOSAPENTAENAMIDE, N-(2,3,3A,9A-TETRAHYDRO-3-HYDROXY-2-(HYDROXYMETHYL)-6H-FURO(2',3':4,5)OXAZOLO(3,2-A)PYRIMIDIN-6-YLIDENE)-, (2R-(2A,3B,3AB,9AB))-/CN

E2 1 EICOSAPENTAENE/CN

E3 3 --> EICOSAPENTAENOIC ACID/CN

E4 1 EICOSAPENTAENOIC ACID Ω 3-EPOXYGENASE/CN

E5 1 EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2037-AMINO ACID FRAGMENT)/CN

E6 1 EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2625-AMINO ACID FRAGMENT)/CN

E7 1 EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 877-AMINO ACID FRAGMENT)/CN

E8 1 EICOSAPENTAENOIC ACID MAGNESIUM SALT/CN

E9 1 EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)METHYL)-2-(((1-OXOHEXADECYL)OXY)ETHYL) ESTER, (Z,Z,Z,Z,Z)-/CN

E10 1 EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN-4-YL)METHYL ESTER/CN

E11 1 EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER/CN

E12 1 EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6,10,14-HEXADECATETRAENYL ESTER/CN

=> s e3

L14 3 "EICOSAPENTAENOIC ACID"/CN

=> s e8

L15 1 "EICOSAPENTAENOIC ACID MAGNESIUM SALT"/CN

=> s e4

L16 1 "EICOSAPENTAENOIC ACID Ω 3-EPOXYGENASE"/CN

=> s e1

L17 1 "EICOSAPENTAENAMIDE, N-(2,3,3A,9A-TETRAHYDRO-3-HYDROXY-2-(HYDROXYMETHYL)-6H-FURO(2',3':4,5)OXAZOLO(3,2-A)PYRIMIDIN-6-YLIDENE)-, (2R-(2A,3B,3AB,9AB))-"/CN

=> s e9-e12

1 "EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)METHYL)-2-(((1-OXOHEXADECYL)OXY)ETHYL) ESTER, (Z,Z,Z,Z,Z)-"/CN

1 "EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN-4-YL)METHYL ESTER"/CN

1 "EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER"/CN

1 "EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6,10,14-HEXADECATETRAENYL ESTER"/CN

L18 4 ("EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)METHYL)-2-(((1-OXOHEXADECYL)OXY)ETHYL) ESTER, (Z,Z,Z,Z,Z)-"/CN OR "EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN-4-YL)METHYL ESTER"/CN OR "EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER"/CN OR "EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6,10,14-HEXADECATETRAENYL ESTER"/CN)

=>

Connection closed by remote host

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTACES1614

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * Welcome to STN International * * * * *

NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	MAY 01	New CAS web site launched
NEWS	3	MAY 08	CA/CAPLUS Indian patent publication number format defined
NEWS	4	MAY 14	RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS	5	MAY 21	BIOSIS reloaded and enhanced with archival data
NEWS	6	MAY 21	TOXCENTER enhanced with BIOSIS reload
NEWS	7	MAY 21	CA/CAPLUS enhanced with additional kind codes for German patents
NEWS	8	MAY 22	CA/CAPLUS enhanced with IPC reclassification in Japanese patents
NEWS	9	JUN 27	CA/CAPLUS enhanced with pre-1967 CAS Registry Numbers
NEWS	10	JUN 29	STN Viewer now available
NEWS	11	JUN 29	STN Express, Version 8.2, now available
NEWS	12	JUL 02	LEMBASE coverage updated
NEWS	13	JUL 02	LMEDLINE coverage updated
NEWS	14	JUL 02	SCISEARCH enhanced with complete author names
NEWS	15	JUL 02	CHEMCATS accession numbers revised
NEWS	16	JUL 02	CA/CAPLUS enhanced with utility model patents from China
NEWS	17	JUL 16	CAPLUS enhanced with French and German abstracts
NEWS	18	JUL 18	CA/CAPLUS patent coverage enhanced
NEWS	19	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS	20	JUL 30	USGENE now available on STN
NEWS	21	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS	22	AUG 06	BEILSTEIN updated with new compounds
NEWS	23	AUG 06	FSTA enhanced with new thesaurus edition
NEWS	24	AUG 13	CA/CAPLUS enhanced with additional kind codes for granted patents
NEWS	25	AUG 20	CA/CAPLUS enhanced with CAS indexing in pre-1907 records

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 14:09:17 ON 24 AUG 2007

=> d his

(FILE 'HOME' ENTERED AT 14:09:17 ON 24 AUG 2007)

=> fil reg

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'REGISTRY' ENTERED AT 14:09:46 ON 24 AUG 2007

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STRUCTURE FILE UPDATES: 23 AUG 2007 HIGHEST RN 945525-31-5

DICTIONARY FILE UPDATES: 23 AUG 2007 HIGHEST RN 945525-31-5

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TSCA INFORMATION NOW CURRENT THROUGH June 29, 2007

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stngen/stndoc/properties.html>

=> e eicosapentaenoic acid ethyl ester/cn

E1	1	EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2625-AMINO ACID FRAGMENT)/CN
E2	1	EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 877-AMINO ACID FRAGMENT)/CN
E3	0 -->	EICOSAPENTAENOIC ACID ETHYL ESTER/CN
E4	1	EICOSAPENTAENOIC ACID MAGNESIUM SALT/CN
E5	1	EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)METHYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-/CN
E6	1	EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN-4-YL)METHYL ESTER/CN
E7	1	EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER/CN
E8	1	EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6,10,14-HEXADECATETRAENYL ESTER/CN
E9	1	EICOSAPENTAENOIC ACID, (2E,7R,11R)-3,7,11,15-TETRAMETHYL-2-HEXADECENYL ESTER/CN
E10	1	EICOSAPENTAENOIC ACID, (ALL-Z)-/CN

E11 1 EICOSAPENTAENOIC ACID, (ALL-Z)-, COMPD. WITH 2A,2B,2C,2D,2E,
2F,6A,6B,6C,6D,6E,6F-DODECA-O-METHYL-A-CYCLODEXTRIN/CN
E12 7 EICOSAPENTAENOIC ACID, (ALL-Z)-, MIXT. CONTG./CN

=> e eicosapentaenoic acid/cn

E1 1 EICOSAPENTAENAMIDE, N-(2,3,3A,9A-TETRAHYDRO-3-HYDROXY-2-(HYD
ROXYMETHYL)-6H-FURO(2',3':4,5)OXAZOLO(3,2-A)PYRIMIDIN-6-YLID
ENE)-, (2R-(2A,3B,3AB,9AB))-/CN
E2 1 EICOSAPENTAENE/CN
E3 3 --> EICOSAPENTAENOIC ACID/CN
E4 1 EICOSAPENTAENOIC ACID Ω 3-EPOXYGENASE/CN
E5 1 EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZ
YME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2037-AMINO ACID
FRAGMENT)/CN
E6 1 EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZ
YME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2625-AMINO ACID
FRAGMENT)/CN
E7 1 EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZ
YME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 877-AMINO ACID
FRAGMENT)/CN
E8 1 EICOSAPENTAENOIC ACID MAGNESIUM SALT/CN
E9 1 EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)
METHYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-/CN
E10 1 EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOX
OLAN-4-YL)METHYL ESTER/CN
E11 1 EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTE
R/CN
E12 1 EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6
,10,14-HEXADECATETRAENYL ESTER/CN

=> s e3

L1 3 "EICOSAPENTAENOIC ACID"/CN

=> s e9-e11

1 "EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)ME
THYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-"/CN
1 "EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLA
N-4-YL)METHYL ESTER"/CN
1 "EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER"/
CN
L2 3 ("EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)M
ETHYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-"/CN OR
"EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN
-4-YL)METHYL ESTER"/CN OR "EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETH
YL-2,6-OCTADIENYL ESTER"/CN)

=> s e9-e12

1 "EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)ME
THYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-"/CN
1 "EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLA
N-4-YL)METHYL ESTER"/CN
1 "EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER"/
CN
1 "EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6,10
,14-HEXADECATETRAENYL ESTER"/CN
L3 4 ("EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)M
ETHYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-"/CN OR
"EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN
-4-YL)METHYL ESTER"/CN OR "EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETH
YL-2,6-OCTADIENYL ESTER"/CN OR "EICOSAPENTAENOIC ACID, (2E,6E,10E
) -3,7,11,15-TETRAMETHYL-2,6,10,14-HEXADECATETRAENYL ESTER"/CN)

=> s e1

L4 1 "EICOSAPENTAENAMIDE, N-(2,3,3A,9A-TETRAHYDRO-3-HYDROXY-2-(HYDROXYMETHYL)-6H-FURO(2',3':4,5)OXAZOLO(3,2-A)PYRIMIDIN-6-YLIDENE)-, (2R-(2A,3B,3AB,9AB))-"/CN

=> s e4

L5 1 "EICOSAPENTAENOIC ACID Ω 3-EPOXYGENASE"/CN

=> s e8

L6 1 "EICOSAPENTAENOIC ACID MAGNESIUM SALT"/CN

=> d his

(FILE 'HOME' ENTERED AT 14:09:17 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 14:09:46 ON 24 AUG 2007

E EICOSAPENTAENOIC ACID ETHYL ESTER/CN

E EICOSAPENTAENOIC ACID/CN

L1 3 S E3

L2 3 S E9-E11

L3 4 S E9-E12

L4 1 S E1

L5 1 S E4

L6 1 S E8

=> e eicosapentanoic acid/cn

E1 1 EICOSAPENTAENOYL CHLORIDE/CN

E2 1 EICOSAPENTAENOYL CHLORIDE, (ALL-Z)-/CN

E3 0 --> EICOSAPENTANOIC ACID/CN

E4 1 EICOSAPENTAYNOIC ACID/CN

E5 1 EICOSAPHINGA-11-ENINE/CN

E6 1 EICOSAPHINGENINE/CN

E7 1 EICOSAPHOSPHA(5)FULLERANE-C20-IH/CN

E8 1 EICOSAPHOSPHA(5)FULLERANE-C20-IH, COMPD. WITH SODIUM (1:1)/CN

E9 1 EICOSAPHOSPHA(5)FULLERANE-C20-IH, COMPD. WITH SODIUM (1:1), ION(1+)/CN

E10 1 EICOSAPHOSPHA(5)FULLERANE-C20-IH, EICOSAOXIDE/CN

E11 1 EICOSAPHOSPHA(5)FULLERANE-C20-IH, EICOSAOXIDE, COMPD. WITH SODIUM (1:1)/CN

E12 1 EICOSAPHOSPHA(5)FULLERANE-C20-IH, EICOSAOXIDE, COMPD. WITH SODIUM ION (NA1-) (1:1)/CN

=> s e4

L7 1 "EICOSAPENTAYNOIC ACID"/CN

=> d

L7 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN

RN 121139-92-2 REGISTRY

ED Entered STN: 16 Jun 1989

CN Eicosapentaynoic acid (9CI) (CA INDEX NAME)

MF C20 H20 O2

CI IDS

SR CA

LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER

CM 1

CRN 506-30-9

CMF C20 H40 O2

HO₂C (CH₂)₁₈ Me

1 REFERENCES IN FILE CA (1907 TO DATE)

1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> d l1

L1 ANSWER 1 OF 3 REGISTRY COPYRIGHT 2007 ACS on STN

RN 32839-30-8 REGISTRY

ED Entered STN: 16 Nov 1984

CN Eicosapentaenoic acid, (Z,Z,Z,Z,Z) - (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN Eicosapentaenoic acid, (all-Z) -

OTHER NAMES:

CN (all-Z)-Eicosapentaenoic acid

CN cis-Eicosapentaenoic acid

CN Eicosapentaenoic acid

MF C20 H30 O2

CI IDS, COM

LC STN Files: ADISNEWS, AGRICOLA, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT,
CIN, EMBASE, PROMT, TOXCENTER, USPAT2, USPATFULL

CM 1

CRN 506-30-9

CMF C20 H40 O2

HO₂C- (CH₂)₁₈-Me

1547 REFERENCES IN FILE CA (1907 TO DATE)

46 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

1548 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> e icosapentaenoic acid/cn

E1 1 ICOSANOYL-COENZYME A SYNTHASE/CN

E2 1 ICOSAPENT/CN

E3 1 --> ICOSAPENTAENOIC ACID/CN

E4 1 ICOSHINE/CN

E5 1 ICOSOLAR T 2357/CN

E6 1 ICOSOLAR W/W 2116/CN

E7 1 ICOSPIRAMIDE/CN

E8 1 ICOSYL D-GLUCOSIDE/CN

E9 1 ICOTEX 520-5016/CN

E10 1 ICOTIDINE/CN

E11 1 ICOTINIC ACID MONONUCLEOTIDE ADENYLYLTRANSFERASE (PSEUDOMONA
S AERUGINOSA STRAIN UCBPP-PA14)/CN

E12 1 ICP 0503/CN

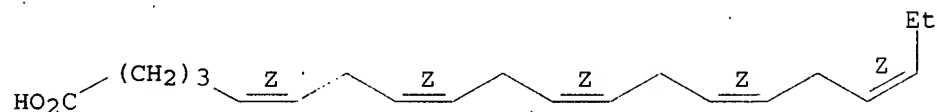
=> s e3

L8 1 "ICOSAPENTAENOIC ACID"/CN

=> d

L8 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
 RN 10417-94-4 REGISTRY
 ED Entered STN: 16 Nov 1984
 CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z) - (CA INDEX NAME)
 OTHER CA INDEX NAMES:
 CN 5,8,11,14,17-Eicosapentaenoic acid (6CI)
 CN 5,8,11,14,17-Eicosapentaenoic acid, (all-Z) - (8CI)
 OTHER NAMES:
 CN (5Z,8Z,11Z,14Z,17Z)-Eicosapentaenoic acid
 CN (all-cis)-5,8,11,14,17-Eicosapentaenoic acid
 CN (all-Z)-Δ5,8,11,14,17-Eicosapentaenoic acid
 CN (all-Z)-5,8,11,14,17-Eicosapentaenoic acid
 CN Eicosapentaenoic acid
 CN EPA
 CN EPA 45G
 CN Icosapent
 CN Icosapentaenoic acid
 CN Incromega E 7010SR
 CN Timnodonic acid
 FS STEREOSEARCH
 DR 25377-48-4
 MF C20 H30 O2
 CI COM
 LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO,
 CA, CABA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CIN, CSCHEM,
 DDFU, DRUGU, EMBASE, IFICDB, IFIUDB, IMSDRUGNEWS, IMSRESEARCH, MRCK*,
 PATDPASPC, PHAR, PROMT, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
 (*File contains numerically searchable property data)
 Other Sources: WHO

Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

10613 REFERENCES IN FILE CA (1907 TO DATE)
 228 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
 10651 REFERENCES IN FILE CAPLUS (1907 TO DATE)
 6 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> d his

(FILE 'HOME' ENTERED AT 14:09:17 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 14:09:46 ON 24 AUG 2007

E EICOSAPENTAENOIC ACID ETHYL ESTER/CN
 E EICOSAPENTAENOIC ACID/CN

L1	3 S E3
L2	3 S E9-E11
L3	4 S E9-E12
L4	1 S E1
L5	1 S E4
L6	1 S E8

E EICOSAPENTANOIC ACID/CN

L7 1 S E4
E ICOSAPENTAENOIC ACID/CN
L8 1 S E3

=> s 11-18

L9 11 (L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8)

=> fil caplus, biosys, medline, uspatfull,
'BIOSYS' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available. If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.

ENTER A FILE NAME OR (IGNORE):biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	75.60	75.81

FILE 'CAPLUS' ENTERED AT 14:16:53 ON 24 AUG 2007
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2007 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 14:16:53 ON 24 AUG 2007
Copyright (c) 2007 The Thomson Corporation

FILE 'MEDLINE' ENTERED AT 14:16:53 ON 24 AUG 2007

FILE 'USPATFULL' ENTERED AT 14:16:53 ON 24 AUG 2007
CA INDEXING COPYRIGHT (C) 2007 AMERICAN CHEMICAL SOCIETY (ACS)

=> s 19

L10 19031 L9

=> s 110 and anorexia

L11 90 L10 AND ANOREXIA

=> s 110 and bulimia

L12 9 L10 AND BULIMIA

=> s 112 or 111

L13 90 L12 OR L11

=> s 112 and pd<07/16/2002

3 FILES SEARCHED...

L14 0 L12 AND PD<07/16/2002

=> s 112 and py<2002

3 FILES SEARCHED...

L15 0 L12 AND PY<2002

=> s 112 and py<2003

3 FILES SEARCHED...

L16 0 L12 AND PY<2003

=> d his

(FILE 'HOME' ENTERED AT 14:09:17 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 14:09:46 ON 24 AUG 2007
E ICOSAPENTAENOIC ACID ETHYL ESTER/CN

```

          E EICOSAPENTAENOIC ACID/CN
L1      3 S E3
L2      3 S E9-E11
L3      4 S E9-E12
L4      1 S E1
L5      1 S E4
L6      1 S E8
          E EICOSAPENTANOIC ACID/CN
L7      1 S E4
          E ICOSAPENTAENOIC ACID/CN
L8      1 S E3
L9      11 S L1-L8

```

FILE 'CAPLUS, BIOSIS, MEDLINE, USPATFULL' ENTERED AT 14:16:53 ON 24 AUG 2007

```

L10      19031 S L9
L11      90 S L10 AND ANOREXIA
L12      9 S L10 AND BULIMIA
L13      90 S L12 OR L11
L14      0 S L12 AND PD<07/16/2002
L15      0 S L12 AND PY<2002
L16      0 S L12 AND PY<2003

```

=> save l1-l16 c10528114/l
L# LIST L1-L16 HAS BEEN SAVED AS 'C10528114/L'

=> l7 and anorexia
L7 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l7 and anorexia
L17 0 L7 AND ANOREXIA

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	27.15	102.96

STN INTERNATIONAL LOGOFF AT 14:29:58 ON 24 AUG 2007

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTACES1614

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * Welcome to STN International * * * * *

NEWS 1 Web Page for STN Seminar Schedule - N. America
NEWS 2 MAY 01 New CAS web site launched
NEWS 3 MAY 08 CA/Capplus Indian patent publication number format defined
NEWS 4 MAY 14 RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS 5 MAY 21 BIOSIS reloaded and enhanced with archival data
NEWS 6 MAY 21 TOXCENTER enhanced with BIOSIS reload
NEWS 7 MAY 21 CA/Capplus enhanced with additional kind codes for German patents
NEWS 8 MAY 22 CA/Capplus enhanced with IPC reclassification in Japanese patents
NEWS 9 JUN 27 CA/Capplus enhanced with pre-1967 CAS Registry Numbers
NEWS 10 JUN 29 STN Viewer now available
NEWS 11 JUN 29 STN Express, Version 8.2, now available
NEWS 12 JUL 02 LEMBASE coverage updated
NEWS 13 JUL 02 LMEDLINE coverage updated
NEWS 14 JUL 02 SCISEARCH enhanced with complete author names
NEWS 15 JUL 02 CHEMCATS accession numbers revised
NEWS 16 JUL 02 CA/Capplus enhanced with utility model patents from China
NEWS 17 JUL 16 Capplus enhanced with French and German abstracts
NEWS 18 JUL 18 CA/Capplus patent coverage enhanced
NEWS 19 JUL 26 USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS 20 JUL 30 USGENE now available on STN
NEWS 21 AUG 06 CAS REGISTRY enhanced with new experimental property tags
NEWS 22 AUG 06 BEILSTEIN updated with new compounds
NEWS 23 AUG 06 FSTA enhanced with new thesaurus edition
NEWS 24 AUG 13 CA/Capplus enhanced with additional kind codes for granted patents
NEWS 25 AUG 20 CA/Capplus enhanced with CAS indexing in pre-1907 records

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

NEWS HOURS STN Operating Hours Plus Help Desk Availability
NEWS LOGIN Welcome Banner and News Items
NEWS IPC8 For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 14:34:58 ON 24 AUG 2007

=> d his

(FILE 'HOME' ENTERED AT 14:34:58 ON 24 AUG 2007)

=> activate c10528114/1

THIS COMMAND NOT AVAILABLE IN THE CURRENT FILE

Some commands only work in certain files. For example, the EXPAND command can only be used to look at the index in a file which has an index. Enter "HELP COMMANDS" at an arrow prompt (=>) for a list of commands which can be used in this file.

=> d save

NAME	CREATED	NOTES/TITLE
CES10763358/L	28 MAR 2007	13 L-NUMBERS
C10484783/L	30 JUL 2007	39 L-NUMBERS
C10489186/L	21 JUN 2007	9 L-NUMBERS
C10497183/L	28 JUN 2007	10 L-NUMBERS
C10500544/L	23 AUG 2007	8 L-NUMBERS
C10528114/L	24 AUG 2007	44 L-NUMBERS
C10575974/L	31 JUL 2007	20 L-NUMBERS
C10727717/L	13 DEC 2006	28 L-NUMBERS
C10814633/L	22 AUG 2007	45 L-NUMBERS
C11021840/L	31 JUL 2007	12 L-NUMBERS
C11084954/L	21 JUN 2007	5 L-NUMBERS
C11312871/L	15 AUG 2007	82 L-NUMBERS
C11641269/L	09 AUG 2007	9 L-NUMBERS
C11641269B/L	TEMP	25 L-NUMBERS
C11641269C/L	13 AUG 2007	59 L-NUMBERS
C11673537/L	02 AUG 2007	48 L-NUMBERS
L10727918/L	09 APR 2007	14 L-NUMBERS
L10803458/L	30 MAR 2007	6 L-NUMBERS
MONO/L	29 JAN 2007	6 L-NUMBERS

=> activate c10528114/1

THIS COMMAND NOT AVAILABLE IN THE CURRENT FILE

Some commands only work in certain files. For example, the EXPAND command can only be used to look at the index in a file which has an index. Enter "HELP COMMANDS" at an arrow prompt (=>) for a list of commands which can be used in this file.

=> help commands

Enter one of these commands at the arrow prompt (=>).

DELETE ----- Delete saved or current session items.
DISPLAY ----- Display saved or current session items.
FILE ----- Specify the search and display file.
HELP ----- For help on how to use the system.
INDEX ----- Specify the Index environment.
LOGOFF ----- End the online session.
NEWS ----- Display current news about the system.
ORDER ----- Order an original document or copy.
SAVE ----- Save an L-numbered query or answer set.
SET ----- Set terminal and interaction options.
? ----- The same as HELP.

Enter one of these commands at the arrow prompt (=>).

For more detailed information about a command, enter HELP followed by the command name at an arrow prompt.

Example: => HELP SEARCH

For information on entering multiple commands at a single arrow prompt, enter HELP COMMAND STACKING.

=> file caplud

'CAPLUD' IS NOT A VALID FILE NAME

SESSION CONTINUES IN FILE 'HOME'

Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.

=> file caplus

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
4.41	4.41

FULL ESTIMATED COST

FILE 'CAPLUS' ENTERED AT 14:47:48 ON 24 AUG 2007

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

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FILE COVERS 1907 - 24 Aug 2007 VOL 147 ISS 10

FILE LAST UPDATED: 23 Aug 2007 (20070823/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply. They are available for your review at:

<http://www.cas.org/infopolicy.html>

=> activate c10528114/1

L1 (3)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	"EICOSAPENTAENOIC ACID"/CN
L2 (3)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	("EICOSAPENTAENOIC ACID, (1R)
L3 (4)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	("EICOSAPENTAENOIC ACID, (1R)
L4 (1)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	"EICOSAPENTAENAMIDE, N-(2,3,3
L5 (1)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	"EICOSAPENTAENOIC ACID .OMEGA
L6 (1)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	"EICOSAPENTAENOIC ACID MAGNES
L7 (1)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	"EICOSAPENTAENOIC ACID"/CN
L8 (1)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	"EICOSAPENTAENOIC ACID"/CN
L9 (11)	SEA FILE=REGISTRY	ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 O
L10 (13284)	SEA FILE=CAPLUS	ABB=ON	PLU=ON	L9
L11 (4820)	SEA FILE=BIOSIS	ABB=ON	PLU=ON	L9
L12 (0)	SEA FILE=MEDLINE	ABB=ON	PLU=ON	L9
L13 (927)	SEA FILE=USPATFULL	ABB=ON	PLU=ON	L9
L14 (19031)	SEA	L9		
L15 (21)	SEA FILE=CAPLUS	ABB=ON	PLU=ON	L10 AND ANOREXIA
L16 (8)	SEA FILE=BIOSIS	ABB=ON	PLU=ON	L11 AND ANOREXIA
L17 (0)	SEA FILE=MEDLINE	ABB=ON	PLU=ON	L12 AND ANOREXIA
L18 (61)	SEA FILE=USPATFULL	ABB=ON	PLU=ON	L13 AND ANOREXIA

L19 (90)SEA L14 AND ANOREXIA
 L20 (2)SEA FILE=CAPLUS ABB=ON PLU=ON L10 AND BULIMIA
 L21 (0)SEA FILE=BIOSIS ABB=ON PLU=ON L11 AND BULIMIA
 L22 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L12 AND BULIMIA
 L23 (7)SEA FILE=USPATFULL ABB=ON PLU=ON L13 AND BULIMIA
 L24 (9)SEA L14 AND BULIMIA
 L25 (21)SEA FILE=CAPLUS ABB=ON PLU=ON L20 OR L15
 L26 (8)SEA FILE=BIOSIS ABB=ON PLU=ON L21 OR L16
 L27 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L22 OR L17
 L28 (61)SEA FILE=USPATFULL ABB=ON PLU=ON L23 OR L18
 L29 (90)SEA L24 OR L19
 L30 (0)SEA FILE=CAPLUS ABB=ON PLU=ON L20 AND PD<07/16/2002
 L31 (0)SEA FILE=BIOSIS ABB=ON PLU=ON L21 AND PD<07/16/2002
 L32 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L22 AND PD<07/16/2002
 L33 (0)SEA FILE=USPATFULL ABB=ON PLU=ON L23 AND PD<07/16/2002
 L34 (0)SEA L24 AND PD<07/16/2002
 L35 (0)SEA FILE=CAPLUS ABB=ON PLU=ON L20 AND PY<2002
 L36 (0)SEA FILE=BIOSIS ABB=ON PLU=ON L21 AND PY<2002
 L37 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L22 AND PY<2002
 L38 (0)SEA FILE=USPATFULL ABB=ON PLU=ON L23 AND PY<2002
 L39 (0)SEA L24 AND PY<2002
 L40 (0)SEA FILE=CAPLUS ABB=ON PLU=ON L20 AND PY<2003
 L41 (0)SEA FILE=BIOSIS ABB=ON PLU=ON L21 AND PY<2003
 L42 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L22 AND PY<2003
 L43 (0)SEA FILE=USPATFULL ABB=ON PLU=ON L23 AND PY<2003
 L44 (0)SEA L24 AND PY<2003

=> s fish oil and (bulimia)

152727 FISH
 10655 FISHES
 155448 FISH
 (FISH OR FISHES)
 788026 OIL
 377937 OILS
 890457 OIL
 (OIL OR OILS)
 10971 FISH OIL
 (FISH(W)OIL)
 1415 BULIMIA
 L45 0 FISH OIL AND (BULIMIA)

=> s fish oil and anorexia

152727 FISH
 10655 FISHES
 155448 FISH
 (FISH OR FISHES)
 788026 OIL
 377937 OILS
 890457 OIL
 (OIL OR OILS)
 10971 FISH OIL
 (FISH(W)OIL)
 7322 ANOREXIA
 8 ANOREXIAS
 7323 ANOREXIA
 (ANOREXIA OR ANOREXIAS)
 L46 13 FISH OIL AND ANOREXIA

=> s fish oil and bulimia

152727 FISH
 10655 FISHES

155448 FISH
(FISH OR FISHES)
788026 OIL
377937 OILS
890457 OIL
(OIL OR OILS)

10971 FISH OIL
(FISH(W)OIL)

1415 BULIMIA

L47 0 FISH OIL AND BULIMIA

=> d scan

L47 HAS NO ANSWERS

=> d scan 145

L45 HAS NO ANSWERS

=> d scan 146

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 4

TI Feeding conjugated linoleic acid to animals partially overcomes catabolic responses due to endotoxin injection

ST linoleate conjugated diet endotoxin growth suppression

IT Animal growth

(endotoxin suppression of, dietary conjugated linoleic acid inhibition of)

IT Toxins

RL: BIOL (Biological study)

(endo-, growth suppression by, dietary conjugated linoleic acid inhibition of)

IT 60-33-3, Linoleic acid, biological studies

RL: BIOL (Biological study)

(growth suppression by endotoxin injection decrease by dietary conjugated)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):5

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN

CC 18-5 (Animal Nutrition)

TI Dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation

ST omega fatty acid sickness behavior inflammation; fish
oil prostaglandin sickness inflammation

IT Rhythm, biological

(circadian; dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Appetite

Blood plasma

Body temperature

Disease, animal

Fever and Hyperthermia

Inflammation

Nutrition, animal

(dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Lipopolysaccharides

Turpentine

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Tumor necrosis factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Fats and Glyceridic oils, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(fish; dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Coconut oil
Coconut oil
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(hydrogenated; dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Fats and Glyceridic oils, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(menhaden; dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Behavior
(motor; dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT Fatty acids, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(polyunsatd., n-3; dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

IT 363-24-6, Prostaglandin E2
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation)

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN

CC 18-5 (Animal Nutrition)

TI Transient hypophagia in rats switched from high-fat diets with different fatty-acid pattern to a high-carbohydrate diet

ST fat carbohydrate diet satiety eating behavior

IT Fats and Glyceridic oils, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(fish; transient hypophagia in response to a change in diet from high-fat to a high-carbohydrate diet)

IT Fatty acids, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(polyunsatd., omega-3; transient hypophagia in response to a change in diet from high-fat to a high-carbohydrate diet)

IT Behavior
Hunger
(transient hypophagia in response to a change in diet from high-fat to a high-carbohydrate diet)

IT Carbohydrates, biological studies
 Fats and Glyceridic oils, biological studies
 Fatty acids, biological studies
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
 (transient hypophagia in response to a change in diet from high-fat to a high-carbohydrate diet)

IT 50-99-7, Glucose, biological studies 300-85-6, β -Hydroxybutyric acid
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (transient hypophagia in response to a change in diet from high-fat to a high-carbohydrate diet)

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN

CC 18-1 (Animal Nutrition)

Section cross-reference(s): 14

TI Dietary patterns of dogs with cardiac disease

ST dog feeding nutrition supplement cardiac disease anorexia

IT Heart, disease

(chronic valvular disease; dietary patterns of dogs with cardiac disease)

IT Anorexia

Canis familiaris

Canned foods

Dietary supplements

Feeding

Nutrition, animal

(dietary patterns of dogs with cardiac disease)

IT Heart, disease

(dilated cardiomyopathy; dietary patterns of dogs with cardiac disease)

IT Food

(sodium-high; dietary patterns of dogs with cardiac disease)

IT Vitamins

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(supplement; dietary patterns of dogs with cardiac disease)

IT 7440-23-5, Sodium, biological studies

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(dietary patterns of dogs with cardiac disease)

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 1

TI Effect of fish oil on appetite and other symptoms in patients with advanced cancer and anorexia/cachexia: a double-blind, placebo, controlled study

ST fish oil appetite cancer anorexia cachexia

eicosapentaenoate docosahexaenoate

IT Cachexia

(cancerous; effect of fish oil on appetite and

other symptoms in cancer patients with anorexia/cachexia)

IT Anorexia

Appetite

Appetite stimulants

Body weight

Fatigue, biological

Human

Neoplasm

Nutrition, animal

(effect of fish oil on appetite and other symptoms in cancer patients with anorexia/cachexia)

IT Fats and Glyceridic oils, biological studies
 RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (fish; effect of fish oil on appetite and other symptoms in cancer patients with anorexia/cachexia)

IT 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 60-33-3, 9,12-Octadecadienoic acid (9Z,12Z)-, biological studies 112-80-1, 9-Octadecenoic acid (9Z)-, biological studies 112-85-6, Docosanoic acid 373-49-9 463-40-1 506-17-2 506-30-9, Eicosanoic acid 506-32-1 544-63-8, Tetradecanoic acid, biological studies 5598-38-9 6217-54-5, DHA 10417-94-4 17046-59-2 20290-75-9 24880-45-3 28874-58-0 28929-01-3 28933-89-3 31152-46-2
 RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (effect of fish oil on appetite and other symptoms in cancer patients with anorexia/cachexia)

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
 CC 1-0 (Pharmacology)
 TI Progress in drug treatment of cancer cachexia
 ST review cancer cachexia drug treatment
 IT Cachexia
 (cancerous; progress in drug treatment of cancer cachexia)
 IT Neoplasm
 (progress in drug treatment of cancer cachexia)
 IT Interleukin 1
 Interleukin 6
 Progestogens
 Tumor necrosis factors
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (progress in drug treatment of cancer cachexia)
 IT 82785-45-3, Neuropeptide Y
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (progress in drug treatment of cancer cachexia)
 IT 50-35-1, Thalidomide 53-03-2, Prednisone 73-31-4, Melatonin 6493-05-6, Pentoxifylline
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (progress in drug treatment of cancer cachexia)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):10

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
 CC 18-5 (Animal Nutrition)
 TI Influence of butter and of corn, coconut and fish oils on the effects of recombinant human tumor necrosis factor- α in rats
 ST fat diet tumor necrosis factor activity; protein metab tumor necrosis factor fat; zinc metab tumor necrosis factor fat
 IT Translation; genetic
 (tumor necrosis factor- α and dietary fat effect on)
 IT Kidney, metabolism
 Liver, metabolism
 Lung, metabolism
 (tumor necrosis factor- α metabolic effects in, dietary fat effect on)
 IT Butter
 Coconut oil
 Corn oil
 Fats and Glyceridic oils
 RL: BIOL (Biological study)

(tumor necrosis factor- α metabolic effects response to dietary)

IT Fats and Glyceridic oils
 RL: BIOL (Biological study)
 (fish, tumor necrosis factor- α metabolic effects response to dietary)

IT Fatty acids, biological studies
 RL: BIOL (Biological study)
 (polyunsatd., n-3, tumor necrosis factor- α metabolic effects response to dietary)

IT Fatty acids, biological studies
 RL: BIOL (Biological study)
 (polyunsatd., n-6, tumor necrosis factor- α metabolic effects response to dietary)

IT Fatty acids, biological studies
 RL: BIOL (Biological study)
 (saturated, tumor necrosis factor- α metabolic effects response to dietary)

IT Lymphokines and Cytokines
 RL: PRP (Properties)
 (tumor necrosis factor- α , metabolic effects of, dietary fat effect on)

IT 7440-66-6, Zinc, biological studies
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (metabolism of, tumor necrosis factor- α and dietary fat effect on)

IT 9031-37-2, Ceruloplasmin 80295-41-6, Complement C 3
 RL: BIOL (Biological study)
 (tumor necrosis factor- α and dietary fat effect on)

IT 60-33-3, Linoleic acid, biological studies
 RL: BIOL (Biological study)
 (tumor necrosis factor- α metabolic effects response to dietary)

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CC 18-5 (Animal Nutrition)
 Section cross-reference(s): 4, 14

TI Effects of dietary n-3 fatty acid supplementation in men with weight loss associated with the acquired immune deficiency syndrome: Relation to indexes of cytokine production.

ST n3 fatty acid diet AIDS wt; fish oil cytokine AIDS body wt

IT Liver
 (liver lipogenesis and cytokine production response to dietary n-3 fatty acid-high fish oil in men with AIDS and weight losses)

IT Glycerides, biological studies
 Lipids, biological studies
 RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (liver lipogenesis and cytokine production response to dietary n-3 fatty acid-high fish oil in men with AIDS and weight losses)

IT Blood
 (nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and blood indexes in men with AIDS and weight losses)

IT Acquired immune deficiency syndrome
 Animal metabolism
 Appetite
 Body weight
 (nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)

- IT Lymphokines and Cytokines
 RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Blood serum
 (nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and serum cytokines in men with AIDS and weight losses)
- IT Fats and Glyceridic oils
 RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
 (fish, MaxEPA; nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Lymphokines and Cytokines
 RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (interleukin 1, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Leukocyte
 (mononuclear, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production by mononuclear cells in men with AIDS and weight losses)
- IT Fatty acids, biological studies
 RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
 (polyunsatd., n-3, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Lymphokines and Cytokines
 RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (tumor necrosis factor, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
- CC 18-0 (Animal Nutrition)
 Section cross-reference(s): 14
- TI Polyunsaturated fatty acids and disease-associated and cytokine-induced neurological manifestations
- ST review nutrition fish oil polyunsatd fatty acid neurol disease
- IT Nutrition, animal
 (dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
- IT Nervous system
 (disease; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
- IT Fats and Glyceridic oils, biological studies
 RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
 (fish; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
- IT Fatty acids, biological studies

RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(polyunsatd.; dietary fish oil and polyunsatd.
fatty acids effects on disease-associated and cytokine-induced neurol.
manifestations)

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
CC 18-5 (Animal Nutrition)
Section cross-reference(s): 1, 14
TI Suppression of tumor growth and metastasis by dietary fish oil combined with vitamins E and C and cisplatin
ST nutrition fish oil vitamin C E antitumor treatment
cisplatin
IT Lung, neoplasm
(carcinoma; dietary fish oil combined with vitamins
E and C and cisplatin effects on suppression of Lewis lung carcinoma
tumor growth and metastasis in mice)
IT Nutrition, animal
(dietary fish oil combined with vitamins E and C
and cisplatin effects on suppression of Lewis lung carcinoma tumor
growth and metastasis in mice)
IT Fats and Glyceridic oils, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); FFD (Food or feed use); BIOL (Biological study);
USES (Uses)
(fish; dietary fish oil combined with vitamins E
and C and cisplatin effects on suppression of Lewis lung carcinoma
tumor growth and metastasis in mice)
IT Antitumor agents
(metastasis; dietary fish oil combined with
vitamins E and C and cisplatin effects on suppression of Lewis lung
carcinoma tumor growth and metastasis in mice)
IT Fatty acids, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); FFD (Food or feed use); BIOL (Biological study);
USES (Uses)
(polyunsatd., n-3; dietary fish oil combined with
vitamins E and C and cisplatin effects on suppression of Lewis lung
carcinoma tumor growth and metastasis in mice)
IT 15663-27-1, Cisplatin
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); BIOL (Biological study)
(Abiplatin; dietary fish oil combined with vitamins
E and C and cisplatin effects on suppression of Lewis lung carcinoma
tumor growth and metastasis in mice)
IT 50-81-7, Vitamin c, biological studies 58-95-7, α -Tocopherol
acetate
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); FFD (Food or feed use); BIOL (Biological study);
USES (Uses)
(dietary fish oil combined with vitamins E and C
and cisplatin effects on suppression of Lewis lung carcinoma tumor
growth and metastasis in mice)

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
CC 18-0 (Animal Nutrition)
Section cross-reference(s): 14
TI Fish oil supplementation in the treatment of cachexia
in pancreatic cancer patients
ST review nutrition fish oil polyunsatd fatty acid
pancreas cancer
IT Cachexia

Human

Nutrition, animal

Pancreas, neoplasm

(dietary fish oil/n-3 polyunsatd. fatty acids

supplementation in treatment of cachexia in pancreatic cancer patients)

IT Fats and Glyceridic oils, biological studies

RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)

(fish; dietary fish oil/n-3 polyunsatd. fatty acids

supplementation in treatment of cachexia in pancreatic cancer patients)

IT Fatty acids, biological studies

RL: BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); USES (Uses)

(polyunsatd., omega-3; dietary fish oil/n-3

polyunsatd. fatty acids supplementation in treatment of cachexia in pancreatic cancer patients)

L46 13 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN

CC 1-0 (Pharmacology)

TI Systemic inflammation, cachexia and prognosis in patients with cancer

ST review cachexia inflammation prognosis cancer eicosapentaenoic acid cyclooxygenase

IT Cachexia

Human

Inflammation

Neoplasm

Prognosis

(systemic inflammation is implicated in hypermetabolism, weight loss associated with cachexia, linked with adverse prognosis and may be important area for novel therapeutic target in combating cachexia in cancer patient)

IT 329900-75-6, Cyclo-oxygenase 2

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(cyclo-oxygenase 2 inhibitors may promote weight gain, downregulate acute phase protein response and appear to be effective against cachexia in cancer patient)

IT 10417-94-4, Eicosapentaenoic acid

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(eicosapentaenoic acid may promote weight gain, downregulate acute phase protein response and appear to be effective against cachexia in cancer patient)

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CC 15-5 (Immunochemistry)

Section cross-reference(s): 2

TI Interleukin-1-induced anorexia in the rat. Influence of prostaglandins

ST interleukin 1 anorexia prostaglandin

IT Corn oil

RL: BIOL (Biological study)

(anorexia induction by interleukin 1 response to feeding with)

IT Prostaglandins

RL: BIOL (Biological study)

(in anorexia induction by interleukin 1)

IT Body weight

(interleukin 1 effect on, in anorexia)

IT Senescence

(interleukin 1-induced anorexia in)

IT Appetite

(disorder, anorexia, interleukin 1-induced, prostaglandin in)

IT Oils, glyceridic
 RL: BIOL (Biological study)
 (fish, anorexia induction by interleukin 1 response to
 feeding with)

IT Lymphokines and Cytokines
 RL: BIOL (Biological study)
 (interleukin 1 α , anorexia induction by, prostaglandins
 in)

IT Lymphokines and Cytokines
 RL: BIOL (Biological study)
 (interleukin 1 β , anorexia induction by, prostaglandins
 in)

IT 60-33-3, Linoleic acid, biological studies 32839-18-2 32839-30-8,
 Eicosapentaenoic acid
 RL: BIOL (Biological study)
 (anorexia induction by interleukin 1 response to feeding
 with)

IT 363-24-6, PGE2
 RL: BIOL (Biological study)
 (in anorexia induction by interleukin 1)

ALL ANSWERS HAVE BEEN SCANNED

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(FILE 'HOME' ENTERED AT 14:34:58 ON 24 AUG 2007)

FILE 'CAPLUS' ENTERED AT 14:47:48 ON 24 AUG 2007

ACTIVATE C10528114/L

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L28 (     61)SEA FILE=USPATFULL ABB=ON  PLU=ON  L23 OR L18
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 L44 (0)SEA L24 AND PY<2003

 E BULIMIA+ALL/CT

L45 0 S FISH OIL AND (BULIMIA)
 L46 13 S FISH OIL AND ANOREXIA
 L47 0 S FISH OIL AND BULIMIA

=> s l46 py<2003

MISSING OPERATOR L46 PY<2003

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=> s l46 and py<2003

22882612 PY<2003

L48 9 L46 AND PY<2003

=> s l46 not l48

L49 4 L46 NOT L48

=> d l48 abs ibib hit kwic

L48 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

AB A study was conducted to determine the prevalence of anorexia and the dietary patterns of dogs with cardiac disease. Sixty-five dogs with cardiac disease were included in the study. Of the 65 dogs, 52 had chronic valvular disease (CVD) and 13 had dilated cardiomyopathy (DCM). There was no difference in diet type between dogs with DCM and CVD but there was a trend for symptomatic dogs to be more likely to eat canned food alone compared to asymptomatic dogs. Seventeen dogs were eating therapeutic diets, although only one was a diet designed specifically for cardiac disease. Sixty of the 65 dogs received treats. Nutritional supplementation was more common in the DCM group than in the CVD group. The most commonly administered nutritional supplements included multivitamin supplements, coenzyme Q10, L-carnitine, taurine, fish oil and vitamin E. Of the 58 owners that administered either medications or nutritional supplements, 36 used human or pet foods for pill administration. Many of these foods were high sodium foods such as lunch meats or cheese.

ACCESSION NUMBER: 2002:466908. CAPLUS

DOCUMENT NUMBER: 138:72414

TITLE: Dietary patterns of dogs with cardiac disease

AUTHOR(S): Freeman, Lisa M.; Rush, John E.; Cahalane, Alane K.; Markwell, Peter J.

CORPORATE SOURCE: Department of Clinical Sciences, Tufts University School of Veterinary Medicine, North Grafton, MA, USA

SOURCE: Journal of Nutrition (2002), 132(6S-2),

1632S-1633S

CODEN: JONUAI; ISSN: 0022-3166

PUBLISHER: American Society for Nutritional Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

SO Journal of Nutrition (2002), 132(6S-2), 1632S-1633S
CODEN: JONUAI; ISSN: 0022-3166

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ST dog feeding nutrition supplement cardiac disease anorexia

IT Anorexia

Canis familiaris

Canned foods

Dietary supplements

Feeding

Nutrition, animal

(dietary patterns of dogs with cardiac disease)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

SO Journal of Nutrition (2002), 132(6S-2), 1632S-1633S
CODEN: JONUAI; ISSN: 0022-3166

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L48 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

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PUBLISHER: American Society for Nutritional Sciences

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CODEN: JONUAI; ISSN: 0022-3166

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ST dog feeding nutrition supplement cardiac disease anorexia

IT Anorexia

Canis familiaris

Canned foods

Dietary supplements

Feeding

Nutrition, animal

(dietary patterns of dogs with cardiac disease)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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CODEN: JONUAI; ISSN: 0022-3166

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ST dog feeding nutrition supplement cardiac disease anorexia

IT Anorexia

Canis familiaris

Canned foods

Dietary supplements

Feeding

Nutrition, animal

(dietary patterns of dogs with cardiac disease)

L48 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

AB The anticancer activity of n-3 polyunsatd. fatty acids (n-3 PUFA) has been shown in a large number of studies. The combined effects of n-3 PUFA and antioxidant vitamins on spontaneous metastatic dissemination were studied in C57BL/6J mice bearing 3LL Lewis lung carcinoma. The supporting effects of this dietary combination on chemotherapy with the conventional cytotoxic agent cisplatin (CP) was also determined. The mice were fed ad libitum 3 isocaloric diets containing 5% soybean oil supplemented with 40 mg α -tocopherol acetate (SO diet)/kg, 4% fish oil plus 1% corn oil plus basal amts. of vitamin E (FO diet), or FO diet supplemented with vitamins E and C (FO+E+C diet). The diets were tested in combination with CP in a series of regimens. The tumor growth, feed consumption, body weight, lung metastasis, and lung histol. were examined. Both FO groups showed lower tumor development than the SO group in all examined parameters, indicating that n-3 PUFA had an anticancer activity. The FO diet, compared to the FO+E+C diet, induced slower rate of tumor growth and decreased the metastatic load as reflected in lung weight. The decrease in the anticancer activity of FO by the addition of vitamins E and C suggested that the in situ oxidation of n-3 PUFA may lead to their anticancer action. Oxidized n-3 PUFA may accumulate in the membranes and cytosol of tumor cells, decrease their vitality, and eventually lead to their death. No signs of anorexia or cachexia were observed in the FO groups, in contrast to the SO group. The CP treatment with the SO diet had no apparent therapeutic effect, while with the FO diets it decreased the metastatic load. The best regimen of this CF-FO combined treatment was the FO diet followed by CP treatment with FO+E+C fed after resection of the primary tumor growth. This regimen could be applicable to combined therapy for human cancer. Thus, diets enriched with n-3 PUFA may have beneficial anticancer effects in particular when containing only basal amts. of antioxidants such as vitamins E or C. The addition of drugs which promote oxidation of n-3 PUFA, such as ferrous salts (as prescribed for the treatment of anemia), may further increase these effects. The supportive effects of n-3 PUFA in chemotherapy with CP increases when vitamins E and C are also included.

ACCESSION NUMBER: 2001:54146 CAPLUS

DOCUMENT NUMBER: 134:310323

TITLE: Suppression of tumor growth and metastasis by dietary

fish oil combined with vitamins E and C and cisplatin

AUTHOR(S): Yam, Daniel; Peled, Alpha; Shinitzky, Meir

CORPORATE SOURCE: Department of Biological Chemistry, Weizmann Institute of Science, Rehovot, 76100, Israel

SOURCE: Cancer Chemotherapy and Pharmacology (2001), 47(1), 34-40
CODEN: CCPHDZ; ISSN: 0344-5704

PUBLISHER: Springer-Verlag

DOCUMENT TYPE: Journal

LANGUAGE: English

TI Suppression of tumor growth and metastasis by dietary fish oil combined with vitamins E and C and cisplatin

SO Cancer Chemotherapy and Pharmacology (2001), 47(1), 34-40
CODEN: CCPHDZ; ISSN: 0344-5704

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ST nutrition fish oil vitamin C E antitumor treatment.
cisplatin

IT Lung, neoplasm
(carcinoma; dietary fish oil combined with vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Nutrition, animal
(dietary fish oil combined with vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Fats and Glyceridic oils, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FED (Food or feed use); BIOL (Biological study);

USES (Uses)

(fish; dietary fish oil combined with vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Antitumor agents

(metastasis; dietary fish oil combined with vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Fatty acids, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study);

USES (Uses)

(polyunsatd., n-3; dietary fish oil combined with vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT 15663-27-1, Cisplatin

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(Abiplatin; dietary fish oil combined with vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT 50-81-7, Vitamin c, biological studies. 58-95-7, α -Tocopherol acetate

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study);

USES (Uses)

(dietary fish oil combined with vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Suppression of tumor growth and metastasis by dietary fish oil combined with vitamins E and C and cisplatin

SO Cancer Chemotherapy and Pharmacology (2001), 47(1), 34-40
CODEN: CCPHDZ; ISSN: 0344-5704

AB The anticancer activity of n-3 polyunsatd. fatty acids (n-3 PUFA) has been shown in a large number of studies. The combined effects of n-3 PUFA and antioxidant vitamins on spontaneous metastatic dissemination were studied in C57BL/6J mice bearing 3LL Lewis lung carcinoma. The supporting effects of this dietary combination on chemotherapy with the conventional cytotoxic agent cisplatin (CP) was also determined. The mice were fed ad libitum 3 isocaloric diets containing 5% soybean oil supplemented with 40 mg α -tocopherol acetate (SO diet)/kg, 4% fish oil plus 1% corn oil plus basal amts. of vitamin E (FO diet), or FO diet supplemented with vitamins E and C (FO+E+C diet). The diets were tested in combination with CP in a series of regimens. The tumor growth, feed consumption, body weight, lung metastasis, and lung histol. were examined. Both FO groups showed lower tumor development than the SO group in all examined parameters, indicating that n-3 PUFA had an anticancer activity. The FO diet, compared to the FO+E+C diet, induced slower rate of tumor growth and decreased the metastatic load as reflected in lung weight. The decrease in the anticancer activity of FO by the addition of vitamins E and C suggested that the in situ oxidation of n-3 PUFA may lead to their anticancer action. Oxidized n-3 PUFA may accumulate in the membranes and cytosol of tumor cells, decrease their vitality, and eventually lead to their death. No signs of anorexia or cachexia were observed in the FO groups, in contrast to the SO group. The CP treatment with the SO diet had no apparent therapeutic effect, while with the FO diets it decreased the metastatic load. The best regimen of this CP-FO combined treatment was the FO diet followed by CP treatment with FO+E+C fed after resection of the primary tumor growth. This regimen could be applicable to combined

therapy for human cancer. Thus, diets enriched with n-3 PUFA may have beneficial anticancer effects in particular when containing only basal amts. of antioxidants such as vitamins E or C. The addition of drugs which promote oxidation of n-3 PUFA, such as ferrous salts (as prescribed for the treatment of anemia), may further increase these effects. The supportive effects of n-3 PUFA in chemotherapy with CP increases when vitamins E and C are also included.

ST nutrition fish oil vitamin C E antitumor treatment

cisplatin

IT Lung, neoplasm

(carcinoma; dietary fish oil combined with vitamins

E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Nutrition, animal

(dietary fish oil combined with vitamins E and C

and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Fats and Glyceridic oils, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); USES (Uses)

(fish; dietary fish oil combined with vitamins E

and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Antitumor agents

(metastasis; dietary fish oil combined with

vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT Fatty acids, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); USES (Uses)

(polyunsatd., n-3; dietary fish oil combined with

vitamins E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT 15663-27-1, Cisplatin

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(Abliplatin; dietary fish oil combined with vitamins

E and C and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

IT 50-81-7, Vitamin c, biological studies 58-95-7, α -Tocopherol acetate

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); USES (Uses)

(dietary fish oil combined with vitamins E and C

and cisplatin effects on suppression of Lewis lung carcinoma tumor growth and metastasis in mice)

L48 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

AB The present study investigates the mechanisms underlying the transient hypophagia occurring when rats adapted to high-fat, carbohydrate-free diets are switched to high-carbohydrate, low-fat diets. The hypophagia after the high-fat, carbohydrate-free to high-carbohydrate, low-fat diet shift seems to depend on the amount of carbohydrate in the diet, since an attenuation of hypophagia was observed when high-fat, carbohydrate-free-adapted rats were switched to a medium-carbohydrate, medium-fat diet. A role of glucose intolerance in the hypophagia is supported by the attenuation of carbohydrate anorexia in rats adapted to a high-fat diet containing n -3 polyunsatd. fatty acids from fish

oil (60% of fat as fish oil), which has been shown to improve glucose tolerance in rats. Furthermore, the increased plasma glucose concentration in the high-fat, carbohydrate-free diet to high-carbohydrate, low-fat shifted rats despite the suppression in food intake also suggests an involvement of glucose intolerance in the hypophagia. The failure of the inhibitor of hepatic-fatty-acid oxidation mercaptoacetate (400 μ mol/kg, i.p.) to counteract carbohydrate anorexia in the HF-adapted rats argues against an involvement of fatty-acids oxidation in the inhibition of eating after high-fat, carbohydrate-free to high-carbohydrate, low-fat diet shift. This is also supported by the failure to demonstrate a relationship between plasma β -hydroxybutyrate and the severity of the hypophagia. A role of leptin in the hypophagia seems unlikely, since plasma leptin after diet shift was unchanged. Ingestion of the high-carbohydrate, low-fat diet also produced an aversion towards this diet in high-fat, carbohydrate-free-adapted rats. It is concluded that the transient hypophagia induced by switching rats from a high-fat to a high-carbohydrate diet is not related to fatty acid oxidation but to transiently impaired carbohydrate utilization. (c) 2000 Academic Press.

ACCESSION NUMBER: 2000:212542 , CAPLUS
DOCUMENT NUMBER: 133:119632
TITLE: Transient hypophagia in rats switched from high-fat diets with different fatty-acid pattern to a high-carbohydrate diet
AUTHOR(S): Del Prete, E.; Lutz, T. A.; Scharrer, E.
CORPORATE SOURCE: Institute of Veterinary Physiology, University of Zurich, Zurich, Switz.
SOURCE: Appetite (London) (2000), 34(2), 137-145
CODEN: APPTD4; ISSN: 0195-6663
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
SO Appetite (London) (2000), 34(2), 137-145
CODEN: APPTD4; ISSN: 0195-6663
AB The present study investigates the mechanisms underlying the transient hypophagia occurring when rats adapted to high-fat, carbohydrate-free diets are switched to high-carbohydrate, low-fat diets. The hypophagia after the high-fat, carbohydrate-free to high-carbohydrate, low-fat diet shift seems to depend on the amount of carbohydrate in the diet, since an attenuation of hypophagia was observed when high-fat, carbohydrate-free-adapted rats were switched to a medium-carbohydrate, medium-fat diet. A role of glucose intolerance in the hypophagia is supported by the attenuation of carbohydrate anorexia in rats adapted to a high-fat diet containing n -3 polyunsatd. fatty acids from fish oil (60% of fat as fish oil), which has been shown to improve glucose tolerance in rats. Furthermore, the increased plasma glucose concentration in the high-fat, carbohydrate-free diet to high-carbohydrate, low-fat shifted rats despite the suppression in food intake also suggests an involvement of glucose intolerance in the hypophagia. The failure of the inhibitor of hepatic-fatty-acid oxidation mercaptoacetate (400 μ mol/kg, i.p.) to counteract carbohydrate anorexia in the HF-adapted rats argues against an involvement of fatty-acids oxidation in the inhibition of eating after high-fat, carbohydrate-free to high-carbohydrate, low-fat diet shift. This is also supported by the failure to demonstrate a relationship between plasma β -hydroxybutyrate and the severity of the hypophagia. A role of leptin in the hypophagia seems unlikely, since plasma leptin after diet shift was unchanged. Ingestion of the high-carbohydrate, low-fat diet also produced an aversion towards this diet in high-fat, carbohydrate-free-adapted rats. It is concluded that the transient hypophagia induced by switching rats from a high-fat to a

high-carbohydrate diet is not related to fatty acid oxidation but to transiently impaired carbohydrate utilization. (c) 2000 Academic Press.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

SO Appetite (London) (2000), 34(2), 137-145
CODEN: APPTD4; ISSN: 0195-6663

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L48 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

AB A review with 63 refs. Dietary fish oil supplementation has been suggested to improve neurol. manifestations of chronic diseases and cytokine immunotherapies. Preclin. and clin. studies show that fish oil supplementation may decrease the disease-associated anorexia and body weight loss. This improvement could be due to shifts in metabolism and changes in proinflammatory cytokine production and action. The ω-3 polyunsatd. fatty acids (PUFAs), especially eicosapentaenoic acid, are substrates in eicosanoid synthesis competing for enzymes with arachidonic acid which is a substrate for the synthesis of proinflammatory immunomodulators, such as PGE2. Fish oil generally lowers the production of cytokines, including interleukin-1 and tumor necrosis factor-α, thereby decreasing various immune responses and inflammation. Conflicting results regarding the effects of fish oil dietary interventions may be due to variety of models, assays, and methodologies used. This review presents an overall perspective on the use of ω-3 PUFAs in nutritional intervention to ameliorate disease-associated and cytokine-induced neurol. symptoms. Substantial further research is required to understand the nature of n-3 PUFA-induced immunomodulation in health and disease.

ACCESSION NUMBER: 1999:434349 CAPLUS

DOCUMENT NUMBER: 131:242374

TITLE: Polyunsaturated fatty acids and disease-associated and cytokine-induced neurological manifestations

AUTHOR(S): Turrin, Nicolas P.; Plata-Salaman, Carlos R.
 CORPORATE SOURCE: Division of Molecular Biology, School of Life and Health Sciences, University of Delaware, Newark, DE, 19716-2590, USA
 SOURCE: Nutritional Neuroscience (1999), 1(6), 395-404
 CODEN: NNINFE; ISSN: 1028-415X
 PUBLISHER: Harwood Academic Publishers
 DOCUMENT TYPE: Journal; General Review
 LANGUAGE: English
 SO Nutritional Neuroscience (1999), 1(6), 395-404
 CODEN: NNINFE; ISSN: 1028-415X
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 ST review nutrition fish oil polyunsatd fatty acid neurol disease
 IT Nutrition, animal
 (dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
 IT Nervous system
 (disease; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
 IT Fats and Glyceridic oils; biological studies
 RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
 (fish; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
 IT Fatty acids, biological studies
 RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
 (polyunsatd.; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
 REFERENCE COUNT: 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
 SO Nutritional Neuroscience (1999), 1(6), 395-404
 CODEN: NNINFE; ISSN: 1028-415X
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- ST review nutrition fish oil polyunsatd fatty acid neurol disease
- IT Nutrition, animal
 - (dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
- IT Nervous system
 - (disease; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
- IT Fats and Glyceridic oils, biological studies
 - RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
 - (fish; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)
- IT Fatty acids, biological studies
 - RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
 - (polyunsatd.; dietary fish oil and polyunsatd. fatty acids effects on disease-associated and cytokine-induced neurol. manifestations)

L48 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

- AB The authors tested the hypothesis that increased dietary fish oil levels (via modulation of the production of inflammatory mediators) modulate sickness symptoms (i.e., anorexia, cachexia, fever, lethargy) of systemic and local inflammation. Swiss Webster mice were implanted with biotelemeters to measure body temperature and motor activity and were fed a diet high in n-3 fatty acids (17% wt/wt menhaden oil) or a reference diet (17% wt/wt hydrogenated coconut oil or normal rodent chow) for 6 wk. Local inflammation was induced by s.c. injection of turpentine (100 μ L/mouse). Systemic inflammation was elicited by i.p. injection of lipopolysaccharide (LPS; 2.5 mg/kg). Fever, lethargy anorexia, and weight decrease during turpentine abscess were all inhibited ($P < 0.05$) in mice fed the fish oil diet. Indomethacin, similar to the fish oil diet, attenuated the turpentine-induced symptoms in mice fed a normal diet. Dietary n-3 fatty acids prevented fever and attenuated the decrease in body weight caused by LPS but did not affect the LPS-induced lethargy and anorexia. Within 90 min of LPS injection, the bioactivity of plasma tumor necrosis factor- α (TNF- α) increased to 98.2 ± 5.1 ng/mL in mice fed fish oil compared with 32.6 ± 3.6 ng/mL in those fed the reference diet ($P < 0.05$). Plasma prostaglandin E₂ (PGE₂) levels after LPS injection of mice fed the control diet increased within 90 min to 16.4 ± 5.1 pg/mL. Mice fed the fish oil diet did not show any elevation in plasma PGE₂ levels at that time ($P < 0.05$). The authors speculate that dietary n-3 fatty acids suppressed PGE₂-related responses, including a PGE₂-dependent neg. feedback on TNF- α production, which resulted in differential modulation of sickness behavior depending on the locus of inflammation.

ACCESSION NUMBER: 1997:300646 CAPLUS
DOCUMENT NUMBER: 127:49839
TITLE: Dietary n-3 fatty acids differentially affect sickness behavior in mice during local and systemic inflammation
AUTHOR(S): Kozak, Wieslaw; Soszynski, Dariusz; Ruldolph, Karin; Conn, Carole A.; Kluger, Matthew J.
CORPORATE SOURCE: Institute for Basic and Applied Medical Research, The Lovelace Institutes, Albuquerque, NM, 87108, USA
SOURCE: American Journal of Physiology (1997), 272(4, Pt. 2), R1298-R1307
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

SO American Journal of Physiology (1997), 272(4, Pt. 2), R1298-R1307

CODEN: AJPHAP; ISSN: 0002-9513

AB The authors tested the hypothesis that increased dietary fish oil levels (via modulation of the production of inflammatory mediators) modulate sickness symptoms (i.e., anorexia, cachexia, fever, lethargy) of systemic and local inflammation. Swiss Webster mice were implanted with biotelemeters to measure body temperature and motor activity and were fed a diet high in n-3 fatty acids (17% wt/wt menhaden oil) or a reference diet (17% wt/wt hydrogenated coconut oil or normal rodent chow) for 6 wk. Local inflammation was induced by s.c. injection of turpentine (100 μ L/mouse). Systemic inflammation was elicited by i.p. injection of lipopolysaccharide (LPS; 2.5 mg/kg). Fever, lethargy anorexia, and weight decrease during turpentine abscess were all inhibited ($P < 0.05$) in mice fed the fish oil diet. Indomethacin, similar to the fish oil diet, attenuated the turpentine-induced symptoms in mice fed a normal diet. Dietary n-3 fatty acids prevented fever and attenuated the decrease in body weight caused by LPS but did not affect the LPS-induced lethargy and anorexia. Within 90 min of LPS injection, the bioactivity of plasma tumor necrosis factor- α (TNF- α) increased to 98.2 ± 5.1 ng/mL in mice fed fish oil compared with 32.6 ± 3.6 ng/mL in those fed the reference diet ($P < 0.05$). Plasma prostaglandin E2 (PGE2) levels after LPS injection of mice fed the control diet increased within 90 min to 16.4 ± 5.1 pg/mL. Mice fed the fish oil diet did not show any elevation in plasma PGE2 levels at that time ($P < 0.05$). The authors speculate that dietary n-3 fatty acids suppressed PGE2-related responses, including a PGE2-dependent neg. feedback on TNF- α production, which resulted in differential modulation of sickness behavior depending on the locus of inflammation.

ST omega fatty acid sickness behavior inflammation; fish oil prostaglandin sickness inflammation

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

SO American Journal of Physiology (1997), 272(4, Pt. 2), R1298-R1307

CODEN: AJPHAP; ISSN: 0002-9513

AB The authors tested the hypothesis that increased dietary fish oil levels (via modulation of the production of inflammatory mediators) modulate sickness symptoms (i.e., anorexia, cachexia, fever, lethargy) of systemic and local inflammation. Swiss Webster mice were implanted with biotelemeters to measure body temperature and motor activity and were fed a diet high in n-3 fatty acids (17% wt/wt menhaden oil) or a reference diet (17% wt/wt hydrogenated coconut oil or normal rodent chow) for 6

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ST omega fatty acid sickness behavior inflammation; fish oil prostaglandin sickness inflammation

L48 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

AB Cytokines may be involved in weight loss and disturbances of metabolism associated

with human immunodeficiency virus (HIV) infection. Dietary n-3 fatty acids reduce the production of interleukin-1 (IL-1) and tumor necrosis factor (TNF) by peripheral blood mononuclear cells (PBMC) in normal humans and prevent IL-1 and TNF anorexia in animals. Accordingly, the authors studied the nutritional and metabolic effects of a 10-wk trial of dietary fish oil (MaxEPA, 18 g/day) in men with weight loss due to acquired immune deficiency syndrome (AIDS). Twenty men were enrolled, and 16 completed the 10-wk supplementation period. Prior weight loss was 13.7 kg. Food intake, body composition, blood chemical, serum

cytokine

concns., in vitro production of IL-1 and TNF by PBMC, and clin. course were followed. A subset of subjects underwent stable isotope infusions to measure de novo hepatic lipogenesis (DNL), an in vivo metabolic index that is influenced by cytokine presence and has previously been found to be elevated in AIDS. An unsupplemented group of men with AIDS wasting (10.4 kg weight loss, 13.1% body weight) was monitored for 10 wk as controls. Baseline food intake (2395 kcal/day and 95.1 g protein/day), body weight, percent fat, and fat-free mass were unchanged over the 10-wk supplementation period. Serum triglycerides were reduced in hypertriglyceridemic subjects, confirming compliance with fish oil supplementation and suggesting that their hypertriglyceridemia was at least in part due to overprod. Serum TNF and IL-1 were undetectable before or after fish oil supplementation. Serum interferon α (IFN) was measurable but did not change. In vitro production of IL-1 and TNF by PBMC was markedly reduced both at baseline and after fish oil supplementation in this population, even in the presence of new AIDS complications, compared with normal controls. The metabolic measurement DNL fell and weight was gained (2.1 kg) in subjects who did not develop new AIDS-related complications, but further increases in DNL and further weight loss were observed in subjects who developed a new AIDS complication. No changes in body weight, food intake, serum triglycerides, or DNL were observed in the unsupplemented group. The authors conclude that fish oil is a weak anticytokine agent which is unable to overcome the metabolic and nutritional consequences of acute AIDS-related complications but may exert a clin.

anticytokine effect in stable AIDS patients. Cytokine production by PBMC is not a useful or reliable marker of in vivo cytokine activity in AIDS patients with weight loss. In contrast, an integrative functional index which is sensitive to cytokine presence in tissues (hepatic DNL) correlated with clin. response. These findings are relevant to the design of future studies of more potent anticytokine agents, such as thalidomide.

ACCESSION NUMBER: 1996:225321 CAPLUS
DOCUMENT NUMBER: 124:287996
TITLE: Effects of dietary n-3 fatty acid supplementation in men with weight loss associated with the acquired immune deficiency syndrome: Relation to indexes of cytokine production.
AUTHOR(S): Hellerstein, Marc K.; Wu, Kenneth; McGrath, Michael; Faix, Dennis; George, Dianne; Shackleton, Cedric H. L.; Horn, William; Hoh, Rebecca; Neese, Richard A.
CORPORATE SOURCE: Department Nutritional Sciences, University California, Berkeley, USA
SOURCE: Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology (1996), 11(3), 258-70
CODEN: JDSRET; ISSN: 1077-9450
PUBLISHER: Lippincott-Raven
DOCUMENT TYPE: Journal
LANGUAGE: English

SO Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology (1996), 11(3), 258-70
CODEN: JDSRET; ISSN: 1077-9450

AB Cytokines may be involved in weight loss and disturbances of metabolism associated

with human immunodeficiency virus (HIV) infection. Dietary n-3 fatty acids reduce the production of interleukin-1 (IL-1) and tumor necrosis factor (TNF) by peripheral blood mononuclear cells (PBMC) in normal humans and prevent IL-1 and TNF anorexia in animals. Accordingly, the authors studied the nutritional and metabolic effects of a 10-wk trial of dietary fish oil (MaxEPA, 18 g/day) in men with weight loss due to acquired immune deficiency syndrome (AIDS). Twenty men were enrolled, and 16 completed the 10-wk supplementation period. Prior weight loss was 13.7 kg. Food intake, body composition, blood chemical, serum cytokine

concns., in vitro production of IL-1 and TNF by PBMC, and clin. course were followed. A subset of subjects underwent stable isotope infusions to measure de novo hepatic lipogenesis (DNL), an in vivo metabolic index that is influenced by cytokine presence and has previously been found to be elevated in AIDS. An unsupplemented group of men with AIDS wasting (10.4 kg weight loss, 13.1% body weight) was monitored for 10 wk as controls. Baseline food intake (2395 kcal/day and 95.1 g protein/day), body weight, percent fat, and fat-free mass were unchanged over the 10-wk supplementation period. Serum triglycerides were reduced in hypertriglyceridemic subjects, confirming compliance with fish oil supplementation and suggesting that their hypertriglyceridemia was at least in part due to overprodn. Serum TNF and IL-1 were undetectable before or after fish oil supplementation. Serum interferon α (IFN) was measurable but did not change. In vitro production of IL-1 and TNF by PBMC was markedly reduced both at baseline and after fish oil supplementation in this population, even in the presence of new AIDS complications, compared with normal controls. The metabolic measurement DNL fell and weight was gained (2.1 kg) in subjects who did not develop new AIDS-related complications, but further increases in DNL and further weight loss were observed in subjects who developed a new AIDS complication. No changes in body weight, food intake, serum triglycerides, or DNL were observed in the unsupplemented group. The authors conclude that fish oil is a weak anticytokine

agent which is unable to overcome the metabolic and nutritional consequences of acute AIDS-related complications but may exert a clin. anticytokine effect in stable AIDS patients. Cytokine production by PBMC is not a useful or reliable marker of in vivo cytokine activity in AIDS patients with weight loss. In contrast, an integrative functional index which is sensitive to cytokine presence in tissues (hepatic DNL) correlated with clin. response. These findings are relevant to the design of future studies of more potent anticytokine agents, such as thalidomide.

ST n3 fatty acid diet AIDS wt; fish oil cytokine AIDS
body wt

IT Liver

(liver lipogenesis and cytokine production response to dietary n-3 fatty acid-high fish oil in men with AIDS and weight losses)

IT Glycerides, biological studies

Lipids, biological studies

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(liver lipogenesis and cytokine production response to dietary n-3 fatty acid-high fish oil in men with AIDS and weight losses)

IT Blood

(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and blood indexes in men with AIDS and weight losses)

IT Acquired immune deficiency syndrome

Animal metabolism

Appetite

Body weight

(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)

IT Lymphokines and Cytokines

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)

IT Blood serum

(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and serum cytokines in men with AIDS and weight losses)

IT Fats and Glyceridic oils

RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(fish, MaxEPA; nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)

IT Lymphokines and Cytokines

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(interleukin 1, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)

IT Leukocyte

(mononuclear, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production by mononuclear cells in men with AIDS and weight losses)

IT Fatty acids, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); THU

(Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(polyunsatd., n-3, nutritional and metabolic effects of dietary n-3
fatty acid-high fish oil and cytokines production in
men with AIDS and weight losses)

IT Lymphokines and Cytokines

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological
study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC
(Process)

(tumor necrosis factor, nutritional and metabolic effects of dietary
n-3 fatty acid-high fish oil and cytokines production
in men with AIDS and weight losses)

SO Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology (1996), 11(3), 258-70

CODEN: JDSRET; ISSN: 1077-9450

AB Cytokines may be involved in weight loss and disturbances of metabolism
associated

with human immunodeficiency virus (HIV) infection. Dietary n-3 fatty
acids reduce the production of interleukin-1 (IL-1) and tumor necrosis factor
(TNF) by peripheral blood mononuclear cells (PBMC) in normal humans and
prevent IL-1 and TNF anorexia in animals. Accordingly, the
authors studied the nutritional and metabolic effects of a 10-wk trial of
dietary fish oil (MaxEPA, 18 g/day) in men with weight
loss due to acquired immune deficiency syndrome (AIDS). Twenty men were
enrolled, and 16 completed the 10-wk supplementation period. Prior weight
loss was 13.7 kg. Food intake, body composition, blood chemical, serum

cytokine

concns., in vitro production of IL-1 and TNF by PBMC, and clin. course were
followed. A subset of subjects underwent stable isotope infusions to
measure de novo hepatic lipogenesis (DNL), an in vivo metabolic index that
is influenced by cytokine presence and has previously been found to be
elevated in AIDS. An unsupplemented group of men with AIDS wasting (10.4
kg weight loss, 13.1% body weight) was monitored for 10 wk as controls.
Baseline food intake (2395 kcal/day and 95.1 g protein/day), body weight,
percent fat, and fat-free mass were unchanged over the 10-wk
supplementation period. Serum triglycerides were reduced in
hypertriglyceridemic subjects, confirming compliance with fish
oil supplementation and suggesting that their hypertriglyceridemia
was at least in part due to overprodn. Serum TNF and IL-1 were
undetectable before or after fish oil supplementation.
Serum interferon α (IFN) was measurable but did not change. In
vitro production of IL-1 and TNF by PBMC was markedly reduced both at baseline
and after fish oil supplementation in this population,
even in the presence of new AIDS complications, compared with normal
controls. The metabolic measurement DNL fell and weight was gained (2.1 kg)
in subjects who did not develop new AIDS-related complications, but
further increases in DNL and further weight loss were observed in subjects who
developed a new AIDS complication. No changes in body weight, food intake,
serum triglycerides, or DNL were observed in the unsupplemented group. The
authors conclude that fish oil is a weak anticytokine
agent which is unable to overcome the metabolic and nutritional
consequences of acute AIDS-related complications but may exert a clin.
anticytokine effect in stable AIDS patients. Cytokine production by PBMC is
not a useful or reliable marker of in vivo cytokine activity in AIDS
patients with weight loss. In contrast, an integrative functional index
which is sensitive to cytokine presence in tissues (hepatic DNL)
correlated with clin. response. These findings are relevant to the design
of future studies of more potent anticytokine agents, such as thalidomide.

ST n3 fatty acid diet AIDS wt; fish oil cytokine AIDS
body wt

IT Liver

(liver lipogenesis and cytokine production response to dietary n-3 fatty

- acid-high fish oil in men with AIDS and weight losses)
- IT Glycerides, biological studies
Lipids, biological studies
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(liver lipogenesis and cytokine production response to dietary n-3 fatty acid-high fish oil in men with AIDS and weight losses)
- IT Blood
(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and blood indexes in men with AIDS and weight losses)
- IT Acquired immune deficiency syndrome
Animal metabolism
Appetite
Body weight
(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Lymphokines and Cytokines
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Blood serum
(nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and serum cytokines in men with AIDS and weight losses)
- IT Fats and Glyceridic oils
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(fish, MaxEPA; nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Lymphokines and Cytokines
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(interleukin 1, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Leukocyte
(mononuclear, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production by mononuclear cells in men with AIDS and weight losses)
- IT Fatty acids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(polyunsatd., n-3, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)
- IT Lymphokines and Cytokines
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(tumor necrosis factor, nutritional and metabolic effects of dietary n-3 fatty acid-high fish oil and cytokines production in men with AIDS and weight losses)

L48 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

AB The ability of conjugated linoleic acid to prevent endotoxin-induced growth suppression was examined. Mice fed a basal diet or a diet with 0.5% fish oil lost twice as much body weight after endotoxin injection than mice fed conjugated linoleic acid. By 72 h post injection, mice fed conjugated linoleic acid had body wts. similar to vehicle injected controls; however, body wts. of basal and fish oil fed mice injected with endotoxin were reduced. Conjugated linoleic acid prevented anorexia from endotoxin injection. Splenocyte blastogenesis was increased by conjugated linoleic acid.

ACCESSION NUMBER: 1994:190339 CAPLUS

DOCUMENT NUMBER: 120:190339

TITLE: Feeding conjugated linoleic acid to animals partially overcomes catabolic responses due to endotoxin injection

AUTHOR(S): Miller, C. C.; Park, Y.; Pariza, M. W.; Cook, M. E.

CORPORATE SOURCE: Food Res. Inst., UW Madison, Madison, WI, 53706, USA

SOURCE: Biochemical and Biophysical Research Communications (1994), 198(3), 1107-12

CODEN: BBRCA9; ISSN: 0006-291X

DOCUMENT TYPE: Journal

LANGUAGE: English

SO Biochemical and Biophysical Research Communications (1994), 198(3), 1107-12

CODEN: BBRCA9; ISSN: 0006-291X

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L48 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

AB Tumor necrosis factor- α is produced in response to inflammatory stimuli. Fish oil can suppress the production and actions of cytokines. Little information is available on the effects of other fats on cytokine biol. The effects of fats with a wide range of fatty acid characteristics were compared on the effects of tumor necrosis factor- α on protein and Zn metabolism in rats. Weanling rats were fed for 8 wk on diets containing 10% fat in the form of corn, fish, or coconut oils or butter before an i.p. injection of recombinant human tumor necrosis factor- α was given. Measurements were made 24 h after the injection. In rats fed corn oil, food intake was reduced by 62% and rates of protein synthesis were increased by 86, 32, and 39% in the liver, lung, and kidney, resp. Zn concns. increased by 23% in the liver but decreased by 10% in the kidney. Plasma ceruloplasmin and complement C3 levels

increased by 25% and 28%, resp., and plasma albumin level decreased by 24%. Fish oil prevented the increase in hepatic protein synthesis and changed the response of protein synthesis in lung and kidney to a decrease. Changes in hepatic and renal Zn concns. were prevented. The response of the plasma ceruloplasmin level was unaltered but those of the plasma complement C3 and albumin concns. were prevented. Coconut oil and butter, although similarly low in linoleic acid, differed in their modulatory effects. With the exception of the rise in the plasma complement C3 concentration, all responses were prevented or greatly inhibited

in

rats fed butter. In rats fed coconut oil the increase in liver protein synthesis was reduced but that in the lung and kidney was unaffected.

Changes in hepatic Zn concentration were unaffected but those in renal Zn concentration

were prevented. Fish and coconut oils and butter reduced the intensity of anorexia caused by tumor necrosis factor- α . The extent to which fats rich in (n-3) polyunsaturates or poor in linoleic acid modulate the metabolic response to tumor necrosis factor- α depends upon addnl. fatty acid characteristics.

ACCESSION NUMBER: 1993:211832 CAPLUS

DOCUMENT NUMBER: 118:211832

TITLE: Influence of butter and of corn, coconut and fish oils on the effects of recombinant human tumor necrosis factor- α in rats

AUTHOR(S): Mulrooney, Hilda M.; Grimble, Robert F.

CORPORATE SOURCE: Inst. Hum. Nutr., Univ. of Southampton, Southampton, UK

SOURCE: Clinical Science (1993), 84(1), 105-12

CODEN: CSCIAE; ISSN: 0143-5221

DOCUMENT TYPE: Journal

LANGUAGE: English

TI Influence of butter and of corn, coconut and fish oils on the effects of recombinant human tumor necrosis factor- α in rats

SO Clinical Science (1993), 84(1), 105-12

CODEN: CSCIAE; ISSN: 0143-5221

AB Tumor necrosis factor- α is produced in response to inflammatory stimuli. Fish oil can suppress the production and actions of cytokines. Little information is available on the effects of other fats on cytokine biol. The effects of fats with a wide range of fatty acid characteristics were compared on the effects of tumor necrosis factor- α on protein and Zn metabolism in rats. Weanling rats were fed for 8 wk on diets containing 10% fat in the form of corn, fish, or coconut oils or butter before an i.p. injection of recombinant human tumor necrosis factor- α was given. Measurements were made 24 h after the injection. In rats fed corn oil, food intake was reduced by 62% and rates of protein synthesis were increased by 86, 32, and 39% in the liver, lung, and kidney, resp. Zn concns. increased by 23% in the liver but decreased by 10% in the kidney. Plasma ceruloplasmin and complement C3 levels increased by 25% and 28%, resp., and plasma albumin level decreased by 24%. Fish oil prevented the increase in hepatic protein synthesis and changed the response of protein synthesis in lung and kidney to a decrease. Changes in hepatic and renal Zn concns. were prevented. The response of the plasma ceruloplasmin level was unaltered but those of the plasma complement C3 and albumin concns. were prevented. Coconut oil and butter, although similarly low in linoleic acid, differed in their modulatory effects. With the exception of the rise in the plasma complement C3 concentration, all responses were prevented or greatly inhibited

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L48 ANSWER 9 OF 9 CAPLUS. COPYRIGHT 2007 ACS on STN

AB The anorexia associated with acute and chronic inflammatory or infectious conditions is poorly understood. The anorexigenic effects of interleukin-1 (IL-1) were examined in the rat. Recombinant human (rh) IL-1 β , murine (rm) IL-1 α and to a lesser extent rhIL-1 α reduced food intake at ≥ 4.0 $\mu\text{g/kg}$ i.p. but not at lower doses, in young (200-250 g) meal-fed rats on chow diets. The anorexic effect appears to be mediated by prostaglandins since pretreatment with ibuprofen completely blocked it, and a fish oil based diet abolished it, in comparison to corn oil or chow diets. Fish oil feeding also decreased basal and IL-1 stimulated prostaglandin E2 production by tissues in vitro (liver, brain, peritoneal macrophages) and in the whole body. Constant i.v. infusions of lower doses of IL-1 also diminished food intake. Chronic daily administration of IL-1 caused persistent inhibition of food intake for 7-17 d in chow and corn oil fed rats, but had no effect in fish oil fed rats. Old (18-20 mo Fisher 344) rats showed less sensitivity to IL-1 induced

anorexia. In conclusion, IL-1 is anorexigenic in the rat, but this is influenced by the structural form of IL-1, the route and chronicity of administration, the source of dietary fat, and the age of the animal. The ability of prior fat intake to influence the anorexic response to IL-1 represents a novel nutrient-nutrient interaction with potential therapeutic implications.

ACCESSION NUMBER: 1989:495213 CAPLUS
DOCUMENT NUMBER: 111:95213
TITLE: Interleukin-1-induced anorexia in the rat.
Influence of prostaglandins
AUTHOR(S): Hellerstein, Marc K.; Meydani, Simin Nikbin; Meydani, Mohsen; Wu, Ken; Dinarello, Charles A.
CORPORATE SOURCE: Hum. Nutr. Res. Cent. Aging, Tufts Univ., Boston, MA, 02111, USA
SOURCE: Journal of Clinical Investigation (1989), 84(1), 228-35
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Interleukin-1-induced anorexia in the rat. Influence of prostaglandins

SO Journal of Clinical Investigation (1989), 84(1), 228-35
CODEN: JCINAO; ISSN: 0021-9738

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ST interleukin 1 anorexia prostaglandin

IT Corn oil

RL: BIOL (Biological study)
(anorexia induction by interleukin 1 response to feeding with)

IT Prostaglandins

RL: BIOL (Biological study)
(in anorexia induction by interleukin 1)

IT Body weight

(interleukin 1 effect on, in anorexia)

IT Senescence

(interleukin 1-induced anorexia in)

IT Appetite

(disorder, anorexia, interleukin 1-induced, prostaglandin in)

IT Oils, glyceridic

RL: BIOL (Biological study)

(fish, anorexia induction by interleukin 1 response to feeding with)

IT Lymphokines and Cytokines
 RL: BIOL (Biological study)
 (interleukin 1 α , anorexia induction by, prostaglandins in)

IT Lymphokines and Cytokines
 RL: BIOL (Biological study)
 (interleukin 1 β , anorexia induction by, prostaglandins in)

IT 60-33-3, Linoleic acid, biological studies 32839-18-2 32839-30-8, Eicosapentaenoic acid
 RL: BIOL (Biological study)
 (anorexia induction by interleukin 1 response to feeding with)

IT 363-24-6, PGE2
 RL: BIOL (Biological study)
 (in anorexia induction by interleukin 1)

TI Interleukin-1-induced anorexia in the rat. Influence of prostaglandins

SO Journal of Clinical Investigation (1989), 84(1), 228-35
 CODEN: JCINAO; ISSN: 0021-9738

AB The anorexia associated with acute and chronic inflammatory or infectious conditions is poorly understood. The anorexigenic effects of interleukin-1 (IL-1) were examined in the rat. Recombinant human (rh) IL-1 β , murine (rm) IL-1 α and to a lesser extent rhIL-1 α reduced food intake at ≥ 4.0 μ g/kg i.p. but not at lower doses, in young (200-250 g) meal-fed rats on chow diets. The anorexic effect appears to be mediated by prostaglandins since pretreatment with ibuprofen completely blocked it, and a fish oil based diet abolished it, in comparison to corn oil or chow diets. Fish oil feeding also decreased basal and IL-1 stimulated prostaglandin E2 production by tissues in vitro (liver, brain, peritoneal macrophages) and in the whole body. Constant i.v. infusions of lower doses of IL-1 also diminished food intake. Chronic daily administration of IL-1 caused persistent inhibition of food intake for 7-17 d in chow and corn oil fed rats, but had no effect in fish oil fed rats. Old (18-20 mo Fisher 344) rats showed less sensitivity to IL-1 induced anorexia. In conclusion, IL-1 is anorexigenic in the rat, but this is influenced by the structural form of IL-1, the route and chronicity of administration, the source of dietary fat, and the age of the animal. The ability of prior fat intake to influence the anorexic response to IL-1 represents a novel nutrient-nutrient interaction with potential therapeutic implications.

ST interleukin 1 anorexia prostaglandin

IT Corn oil
 RL: BIOL (Biological study)
 (anorexia induction by interleukin 1 response to feeding with)

IT Prostaglandins
 RL: BIOL (Biological study)
 (in anorexia induction by interleukin 1)

IT Body weight
 (interleukin 1 effect on, in anorexia)

IT Senescence
 (interleukin 1-induced anorexia in)

IT Appetite
 (disorder, anorexia, interleukin 1-induced, prostaglandin in)

IT Oils, glyceridic
 RL: BIOL (Biological study)
 (fish, anorexia induction by interleukin 1 response to

feeding with)

IT Lymphokines and Cytokines
 RL: BIOL (Biological study)
 (interleukin 1 α , anorexia induction by, prostaglandins
 in)

IT Lymphokines and Cytokines
 RL: BIOL (Biological study)
 (interleukin 1 β , anorexia induction by, prostaglandins
 in)

IT 60-33-3, Linoleic acid, biological studies 32839-18-2 32839-30-8,
 Eicosapentaenoic acid
 RL: BIOL (Biological study)
 (anorexia induction by interleukin 1 response to feeding
 with)

IT 363-24-6, PGE2
 RL: BIOL (Biological study)
 (in anorexia induction by interleukin 1)

=> bulimia

BULIMIA IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.
 For a list of commands available to you in the current file, enter
 "HELP COMMANDS" at an arrow prompt: (=>).

=> d his

(FILE 'HOME' ENTERED AT 14:34:58 ON 24 AUG 2007)

FILE 'CAPLUS' ENTERED AT 14:47:48 ON 24 AUG 2007

ACTIVATE C10528114/L

```

L1 (      3)SEA FILE=REGISTRY ABB=ON  PLU=ON  "EICOSAPENTAENOIC ACID"/CN
L2 (      3)SEA FILE=REGISTRY ABB=ON  PLU=ON  ("EICOSAPENTAENOIC ACID, (1R)
L3 (      4)SEA FILE=REGISTRY ABB=ON  PLU=ON  ("EICOSAPENTAENOIC ACID, (1R)
L4 (      1)SEA FILE=REGISTRY ABB=ON  PLU=ON  "EICOSAPENTAENAMIDE, N-(2,3,3
L5 (      1)SEA FILE=REGISTRY ABB=ON  PLU=ON  "EICOSAPENTAENOIC ACID .OMEGA
L6 (      1)SEA FILE=REGISTRY ABB=ON  PLU=ON  "EICOSAPENTAENOIC ACID MAGNES
L7 (      1)SEA FILE=REGISTRY ABB=ON  PLU=ON  "EICOSAPENTAYNOIC ACID"/CN
L8 (      1)SEA FILE=REGISTRY ABB=ON  PLU=ON  "ICOSAPENTAENOIC ACID"/CN
L9 (     11)SEA FILE=REGISTRY ABB=ON  PLU=ON  (L1 OR L2 OR L3 OR L4 OR L5 O
L10 (    13284)SEA FILE=CAPLUS ABB=ON  PLU=ON  L9
L11 (     4820)SEA FILE=BIOSIS ABB=ON  PLU=ON  L9
L12 (         0)SEA FILE=MEDLINE ABB=ON  PLU=ON  L9
L13 (     927)SEA FILE=USPATFULL ABB=ON  PLU=ON  L9
L14 (    19031)SEA L9
L15 (     21)SEA FILE=CAPLUS ABB=ON  PLU=ON  L10 AND ANOREXIA
L16 (      8)SEA FILE=BIOSIS ABB=ON  PLU=ON  L11 AND ANOREXIA
L17 (      0)SEA FILE=MEDLINE ABB=ON  PLU=ON  L12 AND ANOREXIA
L18 (     61)SEA FILE=USPATFULL ABB=ON  PLU=ON  L13 AND ANOREXIA
L19 (     90)SEA L14 AND ANOREXIA
L20 (      2)SEA FILE=CAPLUS ABB=ON  PLU=ON  L10 AND BULIMIA
L21 (      0)SEA FILE=BIOSIS ABB=ON  PLU=ON  L11 AND BULIMIA
L22 (      0)SEA FILE=MEDLINE ABB=ON  PLU=ON  L12 AND BULIMIA
L23 (      7)SEA FILE=USPATFULL ABB=ON  PLU=ON  L13 AND BULIMIA
L24 (      9)SEA L14 AND BULIMIA
L25 (     21)SEA FILE=CAPLUS ABB=ON  PLU=ON  L20 OR L15
L26 (      8)SEA FILE=BIOSIS ABB=ON  PLU=ON  L21 OR L16
L27 (      0)SEA FILE=MEDLINE ABB=ON  PLU=ON  L22 OR L17
L28 (     61)SEA FILE=USPATFULL ABB=ON  PLU=ON  L23 OR L18
L29 (     90)SEA L24 OR L19

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L30 (0)SEA FILE=CAPLUS ABB=ON PLU=ON L20 AND PD<07/16/2002
 L31 (0)SEA FILE=BIOSIS ABB=ON PLU=ON L21 AND PD<07/16/2002
 L32 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L22 AND PD<07/16/2002
 L33 (0)SEA FILE=USPATFULL ABB=ON PLU=ON L23 AND PD<07/16/2002
 L34 (0)SEA L24 AND PD<07/16/2002
 L35 (0)SEA FILE=CAPLUS ABB=ON PLU=ON L20 AND PY<2002
 L36 (0)SEA FILE=BIOSIS ABB=ON PLU=ON L21 AND PY<2002
 L37 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L22 AND PY<2002
 L38 (0)SEA FILE=USPATFULL ABB=ON PLU=ON L23 AND PY<2002
 L39 (0)SEA L24 AND PY<2002
 L40 (0)SEA FILE=CAPLUS ABB=ON PLU=ON L20 AND PY<2003
 L41 (0)SEA FILE=BIOSIS ABB=ON PLU=ON L21 AND PY<2003
 L42 (0)SEA FILE=MEDLINE ABB=ON PLU=ON L22 AND PY<2003
 L43 (0)SEA FILE=USPATFULL ABB=ON PLU=ON L23 AND PY<2003
 L44 (0)SEA L24 AND PY<2003

 E BULIMIA+ALL/CT

L45 0 S FISH OIL AND (BULIMIA)
 L46 13 S FISH OIL AND ANOREXIA
 L47 0 S FISH OIL AND BULIMIA
 L48 9 S L46 AND PY<2003
 L49 4 S L46 NOT L48

=>

=> save all c10528114/1

'C10528114/L' IN USE

A single name cannot be used for two saved items at the same time.
 Enter "Y" if you wish to replace the current saved name with a new
 definition. Enter "N" if the current saved definition must be
 preserved. You may then reenter the SAVE command with a different
 saved name. Enter "DISPLAY SAVED" at an arrow prompt (=>) to see a
 list of your currently defined saved names.

REPLACE OLD DEFINITION? Y/(N):y

L# LIST L1-L49 HAS BEEN SAVED AS 'C10528114/L'

=> fil stnguide

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	74.28	78.69
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	-7.80	-7.80

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FILE CONTAINS CURRENT INFORMATION.
 LAST RELOADED: Aug 17, 2007 (20070817/UP).

=>

=> file caplus, medline, uspatfull

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	1.98	80.67
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'CAPLUS' ENTERED AT 15:37:31 ON 24 AUG 2007
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FILE 'MEDLINE' ENTERED AT 15:37:31 ON 24 AUG 2007

FILE 'USPATFULL' ENTERED AT 15:37:31 ON 24 AUG 2007
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=> s eicosapentaenoic acid ethyl ester
L50 314 EICOSAPENTAENOIC ACID ETHYL ESTER

=> s l50 and bulimi? OR anorex?
L51 40420 L50 AND BULIMI? OR ANOREX?

=> s l50 and (bulimi? OR anorex?)
L52 4 L50 AND (BULIMI? OR ANOREX?)

=> d 1-4 abs ibib hit kwic

L52 ANSWER 1 OF 4 MEDLINE on STN

AB Anorexia nervosa (AN) carries the highest risk of morbidity and mortality amongst psychiatric disorders. The efficacy of current treatment approaches is limited. Despite the fat-phobic nature of the disease, poly-unsaturated fatty acids (PUFAs) have not received much research attention. Patients who consume western diet, which is rich in n-6 PUFAs and trans-fatty acids, are likely to develop severe n-3 PUFA deficiency during self-induced starvation. Re-feeding programmes do not take into consideration n-3 EPA intake, possibly leading to further n-3 PUFA deficiency during weight restoration, and this might contribute to the maintenance of the disorder. To test this hypothesis, we carried out a systematic case series of E-EPA supplementation in the treatment of AN. Seven young patients received 1g/day E-EPA in addition to standard treatment, and were followed up for 3 months. Three of them recovered and four improved. Randomised controlled trials are warranted to examine the effectiveness of E-EPA in AN further.

ACCESSION NUMBER: 2004398938 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15301789
TITLE: A pilot open case series of ethyl-EPA supplementation in the treatment of anorexia nervosa.
AUTHOR: Ayton Agnes K; Azaz Amer; Horrobin David F
CORPORATE SOURCE: Hull and East Riding Hospitals Trust, UK.
SOURCE: Prostaglandins, leukotrienes, and essential fatty acids, (2004 Oct) Vol. 71, No. 4, pp. 205-9.
Journal code: 8802730. ISSN: 0952-3278.
PUB. COUNTRY: Scotland: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200501
ENTRY DATE: Entered STN: 11 Aug 2004
Last Updated on STN: 29 Jan 2005
Entered Medline: 28 Jan 2005

TI A pilot open case series of ethyl-EPA supplementation in the treatment of anorexia nervosa.

AB Anorexia nervosa (AN) carries the highest risk of morbidity and mortality amongst psychiatric disorders. The efficacy of current treatment approaches is limited. Despite the fat-phobic nature of the disease, poly-unsaturated fatty acids (PUFAs) have not received much

research attention. Patients who consume western diet, which is rich in n-6 PUFAs and trans-fatty acids, are likely to develop severe n-3 PUFA deficiency during self-induced starvation. Re-feeding programmes do not take into consideration n-3 EFA intake, possibly leading to further n-3 PUFA deficiency during weight restoration, and this might contribute to the maintenance of the disorder. To test this hypothesis, we carried out a systematic case series of E-EPA supplementation in the treatment of AN. Seven young patients received 1g/day E-EPA in addition to standard treatment, and were followed up for 3 months. Three of them recovered and four improved. Randomised controlled trials are warranted to examine the effectiveness of E-EPA in AN further.

CT Check Tags: Female; Male

Adolescent

Adult

*Anorexia Nervosa: DT, drug therapy

Anorexia Nervosa: PA, pathology

Eicosapentaenoic Acid: AD, administration & dosage

Eicosapentaenoic Acid: AE, adverse effects

*Eicosapentaenoic Acid: AA, analogs & derivatives

*Eicosapentaenoic Acid: TU, therapeutic use

Follow-Up Studies

Humans

Pilot Projects

Treatment Outcome

RN 1553-41-9 (Eicosapentaenoic Acid); 73310-10-8 (eicosapentaenoic acid ethyl ester)

TI A pilot open case series of ethyl-EPA supplementation in the treatment of anorexia nervosa.

AB Anorexia nervosa (AN) carries the highest risk of morbidity and mortality amongst psychiatric disorders. The efficacy of current treatment approaches is.

CT Check Tags: Female; Male

Adolescent

Adult

*Anorexia Nervosa: DT, drug therapy

Anorexia Nervosa: PA, pathology

Eicosapentaenoic Acid: AD, administration & dosage

Eicosapentaenoic Acid: AE, adverse effects

*Eicosapentaenoic Acid: AA, analogs & . . .

RN 1553-41-9 (Eicosapentaenoic Acid); 73310-10-8 (eicosapentaenoic acid ethyl ester)

L52 ANSWER 2 OF 4 USPATFULL on STN

AB Combinations of omega-3 polyunsaturated fatty acids (PUFAS) and cyclooxygenase-2 selective inhibitors for treatment or prevention of cardiovascular disease, inflammation related disorders or cancer are disclosed. The preferred omega-3 PUFAS of the present invention have from between eighteen and twenty-two carbon atoms, and more preferably from twenty to twenty-two carbon atoms.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2004:58021 USPATFULL

TITLE: Combinations of omega-3 fatty acids and cyclooxygenase-2 inhibitors for treatment or prevention of cardiovascular disease and treatment or prevention of cancer

INVENTOR(S): Obukowicz, Mark G., Kirkwood, MO, UNITED STATES

NUMBER	KIND	DATE
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PATENT INFORMATION:	US 2004044028	A1	20040304
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APPLICATION INFO.: US 2002-113269 AI 20020401 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-280183P	20010330 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Pharmacia Corporation, Corporate Patent Department, 800 N. Lindbergh Blvd., Mail Zone 04E, St. Louis, MO, 63167	
NUMBER OF CLAIMS:	182	
EXEMPLARY CLAIM:	1	
LINE COUNT:	3915	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM [0025] International Application No. 97/39749 describes methods for the prevention and treatment of cachexia and anorexia. Cachexia and anorexia are said to be common conditions among cancer patients whose diseases have progressed to metastatic cancer. The disclosed methods involve administering to an individual an oil blend containing n-6 and omega-3 fatty acids, a source of amino-nitrogen which includes branched-chain amino acids, and an antioxidant component.

DETD [0421] Mice are fed a US 17 diet containing α -linolenic acid ethyl ester (ALA-EE), stearidonic acid ethyl ester (SDA-EE), eicosapentaenoic acid ethyl ester (EPA-EE), or docosahexaenoic acid ethyl ester (DHA-EE) in an amount equivalent to a human western diet containing 1, 3 or 10 g/day of the fatty acid (g/day human equivalent dose). In order to maintain a 17% fat (37% energy) content in the US17 diet, oleic acid (18:1 n-9), as an oleic acid cassette, is removed from the US17 diet in an amount equal to the amount of fatty acid ester that is added. Oleic acid is selected as the replacement fatty acid because literature reports indicate that oleic acid is neutral with respect to inflammation and cancer.

SUMM [0025] International Application No. 97/39749 describes methods for the prevention and treatment of cachexia and anorexia. Cachexia and anorexia are said to be common conditions among cancer patients whose diseases have progressed to metastatic cancer. The disclosed methods involve.

DETD [0421] Mice are fed a US 17 diet containing α -linolenic acid ethyl ester (ALA-EE), stearidonic acid ethyl ester (SDA-EE), eicosapentaenoic acid ethyl ester (EPA-EE), or docosahexaenoic acid ethyl ester (DHA-EE) in an amount equivalent to a human western diet containing 1, 3 or.

L52 ANSWER 3 OF 4 USPATFULL on STN

AB Cancer in a mammal can be treated or prevented by administering to a mammal in need thereof a cancer inhibiting amount of metabolite(s) of α -linolenic acid, such as stearidonic acid (18:4 n-3), eicosatetraenoic acid (20:4 n-3), docosapentaenoic acid (22:5 n-3) and mixtures thereof, especially metabolites including stearidonic acid.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:17331 USPATFULL

TITLE: Use of alpha-linolenic acid metabolites for treatment or prevention of cancer

INVENTOR(S): Obukowicz, Mark, Kirkwood, MO, UNITED STATES
Kabakibi, Ayman, Creve Coeur, MO, UNITED STATES
Green, Susan L., St. Ann, MO, UNITED STATES
Olson, Lisa M., Richmond Heights, MO, UNITED STATES
Lindemann, Julie, El Cerrito, CA, UNITED STATES

NUMBER	KIND	DATE
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PATENT INFORMATION: US 2002010211 A1 20020124
APPLICATION INFO.: US 2001-903707 A1 20010713 (9)
RELATED APPLN. INFO.: Division of Ser. No. US 1999-393790, filed on 10 Sep
1999, UNKNOWN
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FITZPATRICK CELLA HARPER & SCINTO, 30 ROCKEFELLER
PLAZA, NEW YORK, NY, 10112
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 683

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM [0008] International Application No. 97/39749 describes methods for the prevention and treatment of cachexia and anorexia. Cachexia and anorexia are said to be common conditions among cancer patients whose diseases have progressed to metastatic cancer. The disclosed methods involve administering to an individual an oil blend containing n-6 and n-3 fatty acids, a source of amino-nitrogen which includes branched-chain amino acids, and an antioxidant component.

DETD [0055] Mice were fed a US17 diet containing α -linolenic acid ethyl ester (ALA-EE), stearidonic acid ethyl ester (SDA-EE), eicosapentaenoic acid ethyl ester (EPA-EE), or docosahexaenoic acid ethyl ester (DHA-EE) in an amount equivalent to a human western diet containing 1, 3 or 10 g/day of the fatty acid (g/day human equivalent dose). In order to maintain a 17% fat (37 en %) content in the US17 diet, oleic acid (18:1 n-9), as an oleic acid cassette, was removed from the US17 diet in an amount equal to the amount of fatty acid ester that was added. Oleic acid was selected as the replacement fatty acid because literature reports indicate that oleic acid is neutral with respect to inflammation and cancer.

SUMM [0008] International Application No. 97/39749 describes methods for the prevention and treatment of cachexia and anorexia. Cachexia and anorexia are said to be common conditions among cancer patients whose diseases have progressed to metastatic cancer. The disclosed methods involve.

DETD [0055] Mice were fed a US17 diet containing α -linolenic acid ethyl ester (ALA-EE), stearidonic acid ethyl ester (SDA-EE), eicosapentaenoic acid ethyl ester (EPA-EE), or docosahexaenoic acid ethyl ester (DHA-EE) in an amount equivalent to a human western diet containing 1, 3 or.

L52 ANSWER 4 OF 4 USPATFULL on STM

AB Cancer in a mammal can be treated or prevented by administering to a mammal in need thereof a cancer inhibiting amount of metabolite(s) of α -linolenic acid, such as stearidonic acid (18:4 n-3), eicosatetraenoic acid (20:4 n-3), docosapentaenoic acid (22:5 n-3) and mixtures thereof, especially metabolites including stearidonic acid.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:14022 USPATFULL

TITLE: Use of α -linolenic acid metabolites for treatment or prevention of cancer

INVENTOR(S): Obukowicz, Mark, Kirkwood, MO, United States
Kabakibi, Ayman, Creve Coeur, MO, United States
Green, Susan L., St. Ann, MO, United States
Olson, Lisa M., Richmond Heights, MO, United States
Lindemann, Julie, El Cerrito, CA, United States

PATENT ASSIGNEE(S): Monsanto Technology, LLC, St. Louis, MO, United States

(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6340705	B1	20020122
APPLICATION INFO.:	US 1999-393790		19990910 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Criares, Theodore J.		
ASSISTANT EXAMINER:	Kim, J.		
LEGAL REPRESENTATIVE:	Fitzpatrick, Cella, Harper & Scinto		
NUMBER OF CLAIMS:	23		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	681		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM International Application No. 97/39749 describes methods for the prevention and treatment of cachexia and anorexia. Cachexia and anorexia are said to be common conditions among cancer patients whose diseases have progressed to metastatic cancer. The disclosed methods involve administering to an individual an oil blend containing n-6 and n-3 fatty acids, a source of amino-nitrogen which includes branched-chain amino acids, and an antioxidant component.

DETD Mice were fed a US17 diet containing α -linolenic acid ethyl ester (ALA-EE), stearidonic acid ethyl ester (SDA-EE), eicosapentaenoic acid ethyl ester (EPA-EE), or docosahexaenoic acid ethyl ester (DHA-EE) in an amount equivalent to a human western diet containing 1, 3 or 10 g/day of the fatty acid (g/day human equivalent dose). In order to maintain a 17% fat (37 en %) content in the US17 diet, oleic acid (18:1 n-9), as an oleic acid cassette, was removed from the US17 diet in an amount equal to the amount of fatty acid ester that was added. Oleic acid was selected as the replacement fatty acid because literature reports indicate that oleic acid is neutral with respect to inflammation and cancer.

SUMM International Application No. 97/39749 describes methods for the prevention and treatment of cachexia and anorexia. Cachexia and anorexia are said to be common conditions among cancer patients whose diseases have progressed to metastatic cancer. The disclosed methods involve.

DETD Mice were fed a US17 diet containing α -linolenic acid ethyl ester (ALA-EE), stearidonic acid ethyl ester (SDA-EE), eicosapentaenoic acid ethyl ester (EPA-EE), or docosahexaenoic acid ethyl ester (DHA-EE) in an amount equivalent to a human western diet containing 1, 3 or.

=>

=> save all c10528114/1

'C10528114/L' IN USE

A single name cannot be used for two saved items at the same time.

Enter "Y" if you wish to replace the current saved name with a new

definition. Enter "N" if the current saved definition must be

preserved. You may then reenter the SAVE command with a different

saved name. Enter "DISPLAY SAVED" at an arrow prompt (=>) to see a list of your currently defined saved names.

REPLACE OLD DEFINITION? Y/(N):y

L# LIST L1-L52 HAS BEEN SAVED AS 'C10528114/L'

=> save all c10528114/1 y

'Y' IS NOT VALID HERE.

For an explanation, enter "HELP SAVE".

=> save all c10528114/1, y

'Y' IS NOT VALID HERE

For an explanation, enter "HELP SAVE".

=>

=>

=> fil stnguide

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

106.61

187.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

ENTRY

SESSION

CA SUBSCRIBER PRICE

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-7.80

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Connection closed by remote host

---Logging off of STN---

END

Unable to generate the STN prompt.

Exiting the script...

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTACES1614

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * Welcome to STN International * * * * *

NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	MAY 01	New CAS web site launched
NEWS	3	MAY 08	CA/CAPplus Indian patent publication number format defined
NEWS	4	MAY 14	RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS	5	MAY 21	BIOSIS reloaded and enhanced with archival data
NEWS	6	MAY 21	TOXCENTER enhanced with BIOSIS reload
NEWS	7	MAY 21	CA/CAPplus enhanced with additional kind codes for German patents
NEWS	8	MAY 22	CA/CAPplus enhanced with IPC reclassification in Japanese

patents

NEWS 9 JUN 27 CA/CAPLUS enhanced with pre-1967 CAS Registry Numbers

NEWS 10 JUN 29 STN Viewer now available

NEWS 11 JUN 29 STN Express, Version 8.2, now available

NEWS 12 JUL 02 LEMBASE coverage updated

NEWS 13 JUL 02 LEMEDLINE coverage updated

NEWS 14 JUL 02 SCISEARCH enhanced with complete author names

NEWS 15 JUL 02 CHEMCATS accession numbers revised

NEWS 16 JUL 02 CA/CAPLUS enhanced with utility model patents from China

NEWS 17 JUL 16 CAPLUS enhanced with French and German abstracts

NEWS 18 JUL 18 CA/CAPLUS patent coverage enhanced

NEWS 19 JUL 26 USPATFULL/USPAT2 enhanced with IPC reclassification

NEWS 20 JUL 30 USGENE now available on STN

NEWS 21 AUG 06 CAS REGISTRY enhanced with new experimental property tags

NEWS 22 AUG 06 BEILSTEIN updated with new compounds

NEWS 23 AUG 06 PSTA enhanced with new thesaurus edition

NEWS 24 AUG 13 CA/CAPLUS enhanced with additional kind codes for granted patents

NEWS 25 AUG 20 CA/CAPLUS enhanced with CAS indexing in pre-1907 records

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 21:26:39 ON 24 AUG 2007

=> fil reg

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FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 21:26:48 ON 24 AUG 2007

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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 23 AUG 2007 HIGHEST RN 945525-31-5
DICTIONARY FILE UPDATES: 23 AUG 2007 HIGHEST RN 945525-31-5

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TSCA INFORMATION NOW CURRENT THROUGH June 29, 2007

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stngen/stndoc/properties.html>

=> e fish oil/cn

E1	1	FISH MEAL/CN
E2	1	FISH MEAL, HERRING/CN
E3	1 -->	FISH OIL/CN
E4	1	FISH OIL STEARINS/CN
E5	1	FISH OIL, HYDROGENATED/CN
E6	1	FISH OIL, HYDROGENATED, SULFONATED, SODIUM SALTS/CN
E7	1	FISH OIL, OXIDIZED, SULFONATED/CN
E8	1	FISH OIL, POTASSIUM SALT/CN
E9	1	FISH OILS/CN
E10	1	FISH OILS, ANCHOVY/CN
E11	1	FISH OILS, ANCHOVY, SULFATED/CN
E12	1	FISH OILS, BISULFITED/CN

=> s e3

L1 1 "FISH OIL"/CN

=> d

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN

RN 8016-13-5 REGISTRY *

* Use of this CAS Registry Number alone as a search term in other STN files may result in incomplete search results. For additional information, enter HELP RN* at an online arrow prompt (=>).

ED Entered STN: 16 Nov 1984

CN Fish oil (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN Fats and Glyceridic oils, fish

CN Oils, fish

CN Oils, glyceridic, fish

OTHER NAMES:

CN Biopan

CN Defloc Z 3

CN DHA 27C-RD

CN Epamer

CN Eskimo 3

CN Fish oils

CN Glyceridic oils, fish

CN NC 020 (oil)

CN Oils, fish body

CN OmegaPure

CN Pikasol

CN PJ 1

CN Triomar

DEF Extractives and their physically modified derivatives. It consists primarily of the glycerides of C14-C18 and C16-C22 unsatd. fatty acids. (mixed fish).

MF Unspecified

CI COM, MAN, CTS

LC STN Files: ADISNEWS, AGRICOLA, BIOSIS, BIOTECHNO, CA, CAPLUS, CHEMCATS, CHEMLIST, CIN, DETHERM*, EMBASE, HSDB*, IPA, MEDLINE, PATDPASPC, PHAR,

RTECS*, TOXCENTER, USPATFULL

(*File contains numerically searchable property data)

Other Sources: DSL**, EINECS**, TSCA**

(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

4 REFERENCES IN FILE CA (1907 TO DATE)

4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> e eicosapentaenoic acid ethyl ester /cn

E1	1	EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2625-AMINO ACID FRAGMENT)/CN
E2	1	EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 877-AMINO ACID FRAGMENT)/CN
E3	0 -->	EICOSAPENTAENOIC ACID ETHYL ESTER/CN
E4	1	EICOSAPENTAENOIC ACID MAGNESIUM SALT/CN
E5	1	EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)METHYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-/CN
E6	1	EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN-4-YL)METHYL ESTER/CN
E7	1	EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER/CN
E8	1	EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6,10,14-HEXADECATETRAENYL ESTER/CN
E9	1	EICOSAPENTAENOIC ACID, (2E,7R,11R)-3,7,11,15-TETRAMETHYL-2-HEXADECENYL ESTER/CN
E10	1	EICOSAPENTAENOIC ACID, (ALL-Z)-/CN
E11	1	EICOSAPENTAENOIC ACID, (ALL-Z)-, COMPD. WITH 2A,2B,2C,2D,2E,2F,6A,6B,6C,6D,6E,6F-DODECA-O-METHYL-A-CYCLODEXTRIN/CN
E12	7	EICOSAPENTAENOIC ACID, (ALL-Z)-, MIXT. CONTG./CN

=> e eicosapentaenoic acid/cn

E1	1	EICOSAPENTAENAMIDE, N-(2,3,3A,9A-TETRAHYDRO-3-HYDROXY-2-(HYDROXYMETHYL)-6H-FURO(2',3':4,5)OXAZOLO(3,2-A)PYRIMIDIN-6-YLIDENE)-, (2R-(2A,3B,3AB,9AB))-/CN
E2	1	EICOSAPENTAENE/CN
E3	3 -->	EICOSAPENTAENOIC ACID/CN
E4	1	EICOSAPENTAENOIC ACID Ω 3-EPOXYGENASE/CN
E5	1	EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2037-AMINO ACID FRAGMENT)/CN
E6	1	EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 2625-AMINO ACID FRAGMENT)/CN
E7	1	EICOSAPENTAENOIC ACID AND DOCOSAHEXAENOIC ACID SYNTHETIC ENZYME (SHEWANELLA STRAIN SCRC-21406 CLONE 10C7 877-AMINO ACID FRAGMENT)/CN
E8	1	EICOSAPENTAENOIC ACID MAGNESIUM SALT/CN
E9	1	EICOSAPENTAENOIC ACID, (1R)-1-(((BUTOXYHYDROXYPHOSPHINYL)OXY)METHYL)-2-((1-OXOHEXADECYL)OXY)ETHYL ESTER, (Z,Z,Z,Z,Z)-/CN
E10	1	EICOSAPENTAENOIC ACID, (2-METHYL-2-(2-METHYLPROPYL)-1,3-DIOXOLAN-4-YL)METHYL ESTER/CN
E11	1	EICOSAPENTAENOIC ACID, (2E)-3,7-DIMETHYL-2,6-OCTADIENYL ESTER/CN
E12	1	EICOSAPENTAENOIC ACID, (2E,6E,10E)-3,7,11,15-TETRAMETHYL-2,6

10,14-HEXADECATETRAENYL ESTER/CN

=> s e3

L2 3 "EICOSAPENTAENOIC ACID"/CN

=> d

L2 ANSWER 1 OF 3 REGISTRY COPYRIGHT 2007 ACS on STN

RN 32839-30-8 REGISTRY

ED Entered STN: 16 Nov 1984

CN Eicosapentaenoic acid, (Z,Z,Z,Z,Z)- (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN Eicosapentaenoic acid, (all-Z)-

OTHER NAMES:

CN (all-Z)-Eicosapentaenoic acid

CN cis-Eicosapentaenoic acid

CN Eicosapentaenoic acid

MF C20 H30 O2

CI IDS, COM

LC STN Files: ADISNEWS, AGRICOLA, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT,
CIN, EMBASE, PROMT, TOXCENTER, USPAT2, USPATFULL

CM 1

CRN 506-30-9

CMF C20 H40 O2

HO₂C-(CH₂)₁₈-Me

1547 REFERENCES IN FILE CA (1907 TO DATE)

46 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

1548 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> d his

(FILE 'HOME' ENTERED AT 21:26:39 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 21:26:48 ON 24 AUG 2007

E FISH OIL/CN

L1 1 S E3

E EICOSAPENTAENOIC ACID ETHYL ESTER /CN

E EICOSAPENTAENOIC ACID/CN

L2 3 S E3

=> fil embase, uspatful, caplus, medline

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

16.05

16.26

FILE 'EMBASE' ENTERED AT 21:30:01 ON 24 AUG 2007

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FILE 'USPATFULL' ENTERED AT 21:30:01 ON 24 AUG 2007

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FILE 'CAPLUS' ENTERED AT 21:30:01 ON 24 AUG 2007

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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FILE 'MEDLINE' ENTERED AT 21:30:01 ON 24 AUG 2007

=> s horrobin/au (w2) david/au or david/au (w2) horrobin/au
MISSING OPERATOR RROBIN/AU (W2

The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s (horrobin/au (w2) david/au) or (david/au (w2) horrobin/au)
MISSING OPERATOR RROBIN/AU (W2

The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s (horrobin/au (w2) david/au)
MISSING OPERATOR RROBIN/AU (W2

The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s (horrobin/au (w) david/au)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'RROBIN/AU (W) DAVID/AU'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'RROBIN/AU (W) DAVID/AU'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'RROBIN/AU (W) DAVID/AU'
L3 0 (HORROBIN/AU (W) DAVID/AU)

=> s (horrobin/au david/au)
L4 0 (HORROBIN/AU DAVID/AU)

=> s (horrobin/au and david/au)
L5 0 (HORROBIN/AU AND DAVID/AU)

=> s (horrobin/au)
L6 0 (HORROBIN/AU)

=> d his

(FILE 'HOME' ENTERED AT 21:26:39 ON 24 AUG 2007)

FILE 'REGISTRY' ENTERED AT 21:26:48 ON 24 AUG 2007

E FISH OIL/CN
L1 1 S E3
E EICOSAPENTAENOIC ACID ETHYL ESTER /CN
E EICOSAPENTAENOIC ACID/CN
L2 3 S E3

FILE 'EMBASE, USPATFULL, CAPLUS, MEDLINE' ENTERED AT 21:30:01 ON 24 AUG 2007

L3 0 S (HORROBIN/AU (W) DAVID/AU)
L4 0 S (HORROBIN/AU DAVID/AU)
L5 0 S (HORROBIN/AU AND DAVID/AU)
L6 0 S (HORROBIN/AU)

=> s (david/au)
L7 61 (DAVID/AU)

=> s david/au
L8 61 DAVID/AU

=> d scan

L8 61 ANSWERS CAPLUS COPYRIGHT 2007 ACS on STN
IC ICM B01D027-08
ICS B01D035-02
TI Filter and spin on filter. [Machine Translation].

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> s (horrobin (w2) david) or (david (w2) horrobin)
MISSING OPERATOR 'HORROBIN (W2'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s FOGARTY P?/AU
L9 164 FOGARTY P?/AU

=> s horrobin d?/AU
L10 1715 HORROBIN D?/AU

=> s horrobin david?/AU
L11 438 HORROBIN DAVID?/AU

=> s horrobin david?/au
L12 438 HORROBIN DAVID?/AU

=> s l10 and bulimia
L13 2 L10 AND BULIMIA

=> s l10 and anorexia
L14 11 L10 AND ANOREXIA

=> s l10 and cachex?
L15 9 L10 AND CACHEX?

=> s l10 and (l1 or l2)
L16 221 L10 AND (L1 OR L2)

=> s (l13 or l14 or l15) and l16
L17 12 (L13 OR L14 OR L15) AND L16

=> s l17 and py<2002
L18 4 L17 AND PY<2002

=> d scan

L18 4 ANSWERS USPATFULL
AN 2001:86512 USPATFULL
TI Fatty acid esters as bioactive compounds
NCL NCLM: 514/547.000
NCLS: 514/546.000; 514/549.000; 514/552.000; 554/110.000; 554/223.000;
554/224.000; 554/227.000
IC [7]
ICM A61K031-225
IPCI A61K0031-225 [ICM,7]; A61K0031-21 [ICM,7,C*]
IPCR A61K0047-48 [I,C*]; A61K0047-48 [I,A]; A23C [I,S]; A23L [I,S];
A61K [I,S]; A61K0031-21 [I,C*]; A61K0031-23 [I,A]; A61K0031-397
[I,C*]; A61K0031-397 [I,A]; A61K0031-40 [I,C*]; A61K0031-40
[I,A]; A61K0031-403 [I,C*]; A61K0031-403 [I,A]; A61K0031-404
[I,A]; A61K0031-405 [I,A]; A61K0031-415 [I,C*]; A61K0031-415

[I,A]; A61K0031-44 [I,C*]; A61K0031-44 [I,A]; A61K0031-54 [I,C*];
A61K0031-54 [I,A]; A61K0031-5415 [I,C*]; A61K0031-5415 [I,A];
A61K0031-70 [I,C*]; A61K0031-70 [I,A]; A61K0038-22 [I,C*];
A61K0038-22 [I,A]; A61K0038-23 [I,C*]; A61K0038-23 [I,A];
A61K0038-28 [I,C*]; A61K0038-28 [I,A]; A61P0001-00 [I,C*];
A61P0001-00 [I,A]; A61P0003-00 [I,C*]; A61P0003-02 [I,A];
A61P0003-08 [I,A]; A61P0009-00 [I,C*]; A61P0009-08 [I,A];
A61P0009-10 [I,A]; A61P0011-00 [I,C*]; A61P0011-00 [I,A];
A61P0013-00 [I,C*]; A61P0013-02 [I,A]; A61P0015-00 [I,C*];
A61P0015-00 [I,A]; A61P0017-00 [I,C*]; A61P0017-00 [I,A];
A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0025-18 [I,A];
A61P0025-24 [I,A]; A61P0025-26 [I,A]; A61P0025-28 [I,A];
A61P0025-30 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A];
A61P0027-14 [I,A]; A61P0029-00 [I,C*]; A61P0029-00 [I,A];
A61P0035-00 [I,C*]; A61P0035-00 [I,A]; A61P0037-00 [I,C*];
A61P0037-00 [I,A]; A61P0037-08 [I,A]; A61P0043-00 [I,C*];
A61P0043-00 [I,A]; C07C [I,S]; C07C0069-00 [I,C*]; C07C0069-007
[I,A]; C07C0069-58 [I,A]; C07C0069-587 [I,A]; C07C0069-602 [I,A];
C07C0069-76 [I,A]; C07C0229-00 [I,C*]; C07C0229-08 [I,A];
C07C0317-00 [I,C*]; C07C0317-34 [I,A]; C07C0323-00 [I,C*];
C07C0323-52 [I,A]; C07D [I,S]; C07D0207-00 [I,C*]; C07D0207-16
[I,A]; C07D0209-00 [I,C*]; C07D0209-28 [I,A]; C07D0209-40 [I,A];
C07D0213-00 [I,C*]; C07D0213-30 [I,A]; C07D0213-66 [I,A];
C07D0213-67 [I,A]; C07D0213-68 [I,A]; C07D0233-00 [I,C*];
C07D0233-60 [I,A]; C07D0233-94 [I,A]; C07D0279-00 [I,C*];
C07D0279-32 [I,A]; C07D0279-36 [I,A]; C07D0321-00 [I,C*];
C07D0321-00 [I,A]; C07D0339-00 [I,C*]; C07D0339-04 [I,A];
C07D0477-00 [I,C*]; C07D0477-00 [I,A]; C07D0499-00 [I,C*];
C07D0499-00 [I,A]; C07F0009-00 [I,C*]; C07F0009-00 [I,A];
C07F0009-09 [I,A]; C07F0009-655 [I,A]; C07J0001-00 [I,C*];
C07J0001-00 [I,A]; C07J0005-00 [I,C*]; C07J0005-00 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):3

L18 4 ANSWERS USPATFULL

AN 2003:268060 USPATFULL

TI Therapeutic and dietary compositions containing essential fatty acids
and bioactive disulphides

NCL NCLM: 424/439.000

NCLS: 424/464.000; 424/484.000; 424/489.000; 514/724.000; 514/866.000

IC [7]

ICM A61K047-00

IPCI A61K0047-00 [ICM,7]

IPCR A61P0003-00 [I,C*]; A61P0003-10 [I,A]; A61K0031-095 [I,C*];
A61K0031-095 [I,A]; A61K0031-185 [I,C*]; A61K0031-20 [I,A];
A61K0031-202 [I,A]; A61K0031-385 [I,C*]; A61K0031-385 [I,A];
A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0025-00 [I,C*];
A61P0025-00 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A];
A61P0043-00 [I,C*]; A61P0043-00 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L18 4 ANSWERS USPATFULL

AN 2003:115924 USPATFULL

TI 1,3-propane diol esters and ethers and methods for their use in drug
delivery

NCL NCLM: 554/227.000

IC [7]

ICM C07C057-13

IPCI C07C0057-13 [ICM,7]; C07C0057-00 [ICM,7,C*]

IPCR A61K0047-48 [I,C*]; A61K0047-48 [I,A]; A23C [I,S]; A23L [I,S];

A61K [I,S]; A61K0031-21 [I,C*]; A61K0031-23 [I,A]; A61K0031-397 [I,C*]; A61K0031-397 [I,A]; A61K0031-40 [I,C*]; A61K0031-40 [I,A]; A61K0031-403 [I,C*]; A61K0031-403 [I,A]; A61K0031-404 [I,A]; A61K0031-405 [I,A]; A61K0031-415 [I,C*]; A61K0031-415 [I,A]; A61K0031-44 [I,C*]; A61K0031-44 [I,A]; A61K0031-54 [I,C*]; A61K0031-54 [I,A]; A61K0031-5415 [I,C*]; A61K0031-5415 [I,A]; A61K0031-70 [I,C*]; A61K0031-70 [I,A]; A61K0038-22 [I,C*]; A61K0038-22 [I,A]; A61K0038-23 [I,C*]; A61K0038-23 [I,A]; A61K0038-28 [I,C*]; A61K0038-28 [I,A]; A61P0001-00 [I,C*]; A61P0001-00 [I,A]; A61P0003-00 [I,C*]; A61P0003-02 [I,A]; A61P0003-08 [I,A]; A61P0009-00 [I,C*]; A61P0009-08 [I,A]; A61P0009-10 [I,A]; A61P0011-00 [I,C*]; A61P0011-00 [I,A]; A61P0013-00 [I,C*]; A61P0013-02 [I,A]; A61P0015-00 [I,C*]; A61P0015-00 [I,A]; A61P0017-00 [I,C*]; A61P0017-00 [I,A]; A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0025-18 [I,A]; A61P0025-24 [I,A]; A61P0025-26 [I,A]; A61P0025-28 [I,A]; A61P0025-30 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0027-14 [I,A]; A61P0029-00 [I,C*]; A61P0029-00 [I,A]; A61P0035-00 [I,C*]; A61P0035-00 [I,A]; A61P0037-00 [I,C*]; A61P0037-00 [I,A]; A61P0037-08 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07C [I,S]; C07C0069-00 [I,C*]; C07C0069-007 [I,A]; C07C0069-58 [I,A]; C07C0069-587 [I,A]; C07C0069-602 [I,A]; C07C0069-76 [I,A]; C07C0229-00 [I,C*]; C07C0229-08 [I,A]; C07C0317-00 [I,C*]; C07C0317-34 [I,A]; C07C0323-00 [I,C*]; C07C0323-52 [I,A]; C07D [I,S]; C07D0207-00 [I,C*]; C07D0207-16 [I,A]; C07D0209-00 [I,C*]; C07D0209-28 [I,A]; C07D0209-40 [I,A]; C07D0213-00 [I,C*]; C07D0213-30 [I,A]; C07D0213-66 [I,A]; C07D0213-67 [I,A]; C07D0213-68 [I,A]; C07D0233-00 [I,C*]; C07D0233-60 [I,A]; C07D0233-94 [I,A]; C07D0279-00 [I,C*]; C07D0279-32 [I,A]; C07D0279-36 [I,A]; C07D0321-00 [I,C*]; C07D0321-00 [I,A]; C07D0339-00 [I,C*]; C07D0339-04 [I,A]; C07D0477-00 [I,C*]; C07D0477-00 [I,A]; C07D0499-00 [I,C*]; C07D0499-00 [I,A]; C07F0009-00 [I,C*]; C07F0009-00 [I,A]; C07F0009-09 [I,A]; C07F0009-655 [I,A]; C07J0001-00 [I,C*]; C07J0001-00 [I,A]; C07J0005-00 [I,C*]; C07J0005-00 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

L18 4 ANSWERS USPATFULL

AN 2002:144250 USPATFULL

TI Fatty acid treatment

NCL NCLM: 514/034.000

NCLS: 514/560.000

IC [7]

ICM A61K031-70

ICS A61K031-20

IPCI A61K0031-70 [ICM,7]; A61K0031-20 [ICS,7]; A61K0031-185 [ICS,7,C*]

IPCR A61P0035-00 [I,C*]; A61P0035-00 [I,A]; A61K0031-185 [I,C*];

A61K0031-201 [I,A]; A61K0031-202 [I,A]

PAGE IMAGES NOT AVAILABLE FOR THIS PATENT

ALL ANSWERS HAVE BEEN SCANNED

=> d 118 1-4 abs bib hit kwic

L18 ANSWER 1 OF 4 USPATFULL on STN

AB Compositions of GLA and/or other EFAs with TA or related compounds, and their use in therapy or nutrition or in preparation of composition for therapy or nutrition, especially to improve cell membrane EFA concentration and/or (particularly in diabetic complications) impaired nerve function and blood flow.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AN 2003:268060 USPATFULL
TI Therapeutic and dietary compositions containing essential fatty acids and bioactive disulphides
IN Horrobin, David F., Stirling, UNITED KINGDOM
Tritschler, Hans-Jurgen, Bad Homberg, GERMANY, FEDERAL REPUBLIC OF
PA Viatris GmbH & Co. KG., GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)
PI US 6630157 B1 20031007
WO 9904782 19990204
AI US 2000-463300 20000815 (9)
WO 1998-GB2155 19980722
PRAI GB 1997-15444 19970722
DT Utility
FS GRANTED
EXNAM Primary Examiner: Page, Thurman K.; Assistant Examiner: Evans, Charesse
LREP Nixon & Vanderhye P.C.
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 478

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IN Horrobin, David F., Stirling, UNITED KINGDOM
PI US 6630157 B1 20031007
WO 9904782 19990204
SUMM j) any form of cancer or pre-cancerous condition, including cachexia associated with cancer.

IT 506-26-3, γ -Linolenic acid 506-32-1, Arachidonic acid
1200-22-2, α -Lipoic acid 1783-84-2, Dihomo- γ -linolenic acid
6217-54-5, Docosahexaenoic acid 6629-12-5, Tetranorlipoic acid
10417-94-4, Eicosapentaenoic acid 13125-44-5, Bisnorlipoic acid
20290-75-9, Stearidonic acid 32839-34-2, Docosapentaenoic acid
56578-24-6
(therapeutic and dietary compns. containing essential fatty acids and bioactive disulfides)

IN Horrobin, David F., Stirling, UNITED KINGDOM
PI US 6630157 B1 20031007
WO 9904782 19990204

SUMM j) any form of cancer or pre-cancerous condition, including cachexia associated with cancer.

IT 506-26-3, γ -Linolenic acid 506-32-1, Arachidonic acid
1200-22-2, α -Lipoic acid 1783-84-2, Dihomo- γ -linolenic acid
6217-54-5, Docosahexaenoic acid 6629-12-5, Tetranorlipoic acid
10417-94-4, Eicosapentaenoic acid 13125-44-5, Bisnorlipoic acid
20290-75-9, Stearidonic acid 32839-34-2, Docosapentaenoic acid
56578-24-6
(therapeutic and dietary compns. containing essential fatty acids and bioactive disulfides)

L18 ANSWER 2 OF 4 USPATFULL on STN

AB The invention relates to compounds of formula: ##STR1##

wherein R.sup.1 is selected from the group consisting of fatty acid acyl groups of 12 to 30 carbon atoms and fatty alcohol groups of 12 to 30 carbon atoms, and wherein R.sup.2 is selected from the group consisting of H, fatty acid acyl of 12 to 30 carbon atoms and fatty alcohol groups of 12 to 30 carbon atoms, the same as or different from R.sup.1, and the residue of a nutrient, drug, or other bioactive compound, and to the use of these compounds to deliver drugs and other bioactive compounds.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AN 2003:115924 USPATFULL
TI 1,3-propane diol esters and ethers and methods for their use in drug delivery
IN Horrobin, David Frederick, Guildford, UNITED KINGDOM
Manku, Mehar, Carlisle, UNITED KINGDOM
McMordie, Austin, Carlisle, UNITED KINGDOM
Knowles, Philip, Carlisle, UNITED KINGDOM
Redden, Peter, Nova Scotia, CANADA
Pitt, Andrea, Carlisle, UNITED KINGDOM
Bradley, Paul, Carlisle, UNITED KINGDOM
Wakefield, Paul, Carlisle, UNITED KINGDOM
PA Scotia Holdings plc, Stirling, UNITED KINGDOM (non-U.S. corporation)
PI US 6555700 B1 20030429
WO 9634846 19961107
AI US 1998-945667 19980128 (8)
WO 1996-GB1053 19960501
PRAI GB 1995-8823 19950501
GB 1995-17107 19950821
GB 1996-5440 19960315
DT Utility
FS GRANTED
EXNAM Primary Examiner: McKane, Joseph K.; Assistant Examiner: Anderson, Rebecca
LREP Russell, Dean W., Gray, Bruce D., Kilpatrick Stockton LLP
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 0 Drawing Figure(s); 0 Drawing Page(s)
LN.CNT 2165

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IN Horrobin, David Frederick, Guildford, UNITED KINGDOM
PI US 6555700 B1 20030429
WO 9634846 19961107
SUMM 6. Anticancer effects of three sorts, selective cytotoxic damage and induction of apoptosis in cancer cells but not in normal cells, inhibition of growth by reduction of action of growth factors and interference with second messenger systems required for growth, inhibition of metastasis by various actions including increased expression of E-cadherins and inhibition of proteolytic enzymes such as urokinases, lipoxxygenase and matrix metalloproteinases, and inhibition of cancer-associated cachexia.

SUMM (s) cancer cachexia.

IT 53-86-1, Indomethacin 88-82-4, 2,3,5-Triiodobenzoic acid 506-26-3
1077-28-7, 1,2-Dithiolane-3-pentanoic acid 10417-94-4
54562-14-0 98770-65-1
(preparation of fatty acid esters as bioactive compds.)

IN Horrobin, David Frederick, Guildford, UNITED KINGDOM
PI US 6555700 B1 20030429
WO 9634846 19961107

SUMM . . . increased expression of E-cadherins and inhibition of proteolytic enzymes such as urokinases, lipoxxygenase and matrix metalloproteinases, and inhibition of cancer-associated cachexia

SUMM (s) cancer cachexia.

IT 53-86-1, Indomethacin 88-82-4, 2,3,5-Triiodobenzoic acid 506-26-3
1077-28-7, 1,2-Dithiolane-3-pentanoic acid 10417-94-4
54562-14-0 98770-65-1
(preparation of fatty acid esters as bioactive compds.)

L18 ANSWER 3 OF 4 USPATFULL on STN

AB The use in preparation of a medicament for treating and preventing the side effects of anti-cancer chemotherapy of a polyunsaturated fatty acid with a carbon chain length of 14 to 26 and with 2 to 6 double bonds in the molecule in cis or trans configuration, and a method of such treatment or prevention wherein said fatty acid is used as an active.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AN 2002:144250 USPATFULL

TI Fatty acid treatment

IN Scott, Catherine A., c/o 33 Hanbury Close, Cheshunt, Waltham Cross, Herts, UNITED KINGDOM EN8 9BZ

Horrobin, David F., Laxdale Ltd. Kings Park House, Laurelhill Business Park, Stirling, UNITED KINGDOM FK7 9JQ

PI US 6407075 B1 20020618

WO 9809621 19980312

AI US 1999-254286 19990706 (9)

WO 1997-GB2362 19970902

19990706 PCT 371 date

PRAI GB 1996-18420 19960904

DT Utility

FS GRANTED

EXNAM Primary Examiner: Goldberg, Jerome D.

CLMN Number of Claims: 2

ECL Exemplary Claim: 1

DRWN 0 Drawing Figure(s); 0 Drawing Page(s)

LN.CNT 293

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IN Horrobin, David F., Laxdale Ltd. Kings Park House, Laurelhill Business Park, Stirling, UNITED KINGDOM FK7 9JQ

PI US 6407075 B1 20020618

WO 9809621 19980312

DETD A. A patient with malignant brain cancer, a glioblastoma, was treated in addition to surgery with the nitrosourea, carmustine. Prior to and during the carmustine course the patient was also receiving gamma-linolenic acid and linoleic acid in the form of the triglyceride dilinoleoyl-monogammalinolenoyl-glycerol (DLMG). Carmustine normally causes marked nausea and vomiting, anorexia and severe malaise. in contrast, this patient experienced only very mild nausea and felt well throughout indicating reduction of the side effects by the DLMG.

DETD B. A patient with Hodgkin's disease was treated with the 'MOPP' regime (carmustine, vincristine, procarbazine and prednisone). For one week prior to the start of the chemotherapy regime, and throughout the course of chemotherapy, this 40 year old man also received 3 g per day orally of the lithium salt of gammalinolenic acid. This chemotherapy regime normally causes severe vomiting and nausea, marked malaise and anorexia. In contrast, this patient experienced only mild nausea and remained well and able to continue working.

IT 60-33-3, Linoleic acid, biological studies 463-40-1, α -Linolenic acid 506-26-3, γ -Linolenic acid 1783-84-2, Dihomo γ -linolenic acid 6217-54-5, Docosaheptaenoic acid 10417-94-4, Eicosapentaenoic acid 20290-75-9, Stearidonic acid 112076-80-9 122051-95-0, Lithium γ -Linolenate 204708-21-4 (fatty acids for preventing side effects of chemotherapy)

IN Horrobin, David F., Laxdale Ltd. Kings Park House, Laurelhill Business Park, Stirling, UNITED KINGDOM FK7 9JQ

PI US 6407075 B1 20020618

WO 9809621 19980312

DETD gamma-linolenic acid and linoleic acid in the form of the

triglyceride dilinoleoyl-monogammalinolenoyl-glycerol (DLMG). Carmustine normally causes marked nausea and vomiting, anorexia and severe malaise. in contrast, this patient experienced only very mild nausea and felt well throughout indicating reduction of the.

DETD . . . orally of the lithium salt of gammalinolenic acid. This chemotherapy regime normally causes severe vomiting and nausea, marked malaise and anorexia. In contrast, this patient experienced only mild nausea and remained well and able to continue working.

IT 60-33-3, Linoleic acid, biological studies 463-40-1, α -Linolenic acid 506-26-3, γ -Linolenic acid 1783-84-2, Dihomo γ -linolenic acid 6217-54-5, Docosahexaenoic acid 10417-94-4, Eicosapentaenoic acid 20290-75-9, Stearidonic acid. 112076-80-9 122051-95-0, Lithium γ -Linolenate 204708-21-4 (fatty acids for preventing side effects of chemotherapy)

L18 ANSWER 4 OF 4 USPATFULL on STN

AB Compounds of structure (I), and when for use in therapy: where R.sub.1 is an acyl group derived from a C.sub.16-30 fatty acid with two or more cis or trans double bonds and particularly an n-6 or n-3 series EPA or conjugated linoleic acid, or columbinic acid, or parinaric acid and R.sub.2 is as R.sub.1 the same or different, or any other nutrient, drug or other bioactive residue released as the active in the body and R.sub.3 is either hydrogen, fully hydrocarbon, or containing heteroatoms, preferably an alkyl group particularly a C.sub.1 -C.sub.4 alkyl group. ##STR1##

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AN 2001:86512 USPATFULL

TI Fatty acid esters as bioactive compounds

IN Horrobin, David Frederick, Guildford, United Kingdom
Manku, Mehar, Carlisle, United Kingdom
McMordie, Austin, Carlisle, United Kingdom
Knowles, Philip, Carlisle, United Kingdom
Redden, Peter, Nova Scotia, United Kingdom
Pitt, Andrea, Carlisle, United Kingdom

PA Scotia Holdings PLC, Surrey, United Kingdom (non-U.S. corporation)

PI US 6245811 B1 20010612 <--

AI US 1999-376617 19990818 (9)

RLI Division of Ser. No. US 945779, now abandoned

PRAI GB 1995-8823 19950501

GB 1995-17107 19950821

GB 1996-5440 19960315

DT Utility

FS GRANTED

EXNAM Primary Examiner: Carr, Deborah D.

LREP Gray, Esq., Bruce D.Kilpatrick Stockton LLP

CLMN Number of Claims: 64

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 1248

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IN Horrobin, David Frederick, Guildford, United Kingdom

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SUMM 6. Anticancer effects of three sorts, selective cytotoxic damage and induction of apoptosis in cancer cells but not in normal cells, inhibition of growth by reduction of action of growth factors and interference with second messenger systems required for growth, inhibition of metastasis by various actions including increased expression of E-cadherins and inhibition of proteolytic enzymes such as urokinases, lipoxxygenase and matrix metalloproteinases, and inhibition of cancer-associated cachexia.

SUMM (s) cancer cachexia.

CLM What is claimed is:

28. A method for treating a disorder selected from the group consisting of complications of diabetes; cancer; osteoarthritis; rheumatoid arthritis; inflammatory and auto-immune diseases other than arthritis; respiratory diseases; neurological disorders; renal and urinary tract disorders; cardiovascular disorders; degenerative diseases of the eye; psychiatric disorders; prostatic hypertrophy and prostatitis; impotence and male infertility; mastalgia; male pattern baldness; osteoporosis; dermatological disorders; dyslexia and other learning disabilities; and cancer cachexia; comprising administering to a patient in need thereof an effective amount of the compound of claim 10.

37. The method according to claim 28, wherein said disorder is selected from the group consisting of cancer; osteoarthritis; rheumatoid arthritis; inflammatory and auto-immune diseases other than arthritis; respiratory diseases; neurological disorders; renal and urinary tract disorders; cardiovascular disorders; degenerative diseases of the eye; psychiatric disorders; osteoporosis; dermatological disorders; dyslexia and other learning disabilities; and cancer cachexia; and wherein R.sup.1 is eicosapentaenoic acid (EPA) and R.sub.2 is selected from the group consisting of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

64. A method for treating cancer cachexia, comprising administering to a patient in need thereof an effective amount of the compound of claim 10.

IT 53-86-1, Indomethacin 88-82-4, 2,3,5-Triiodobenzoic acid 506-26-3
1077-28-7, 1,2-Dithiolane-3-pentanoic acid 10417-94-4
54562-14-0 98770-65-1

(preparation of fatty acid esters as bioactive compds.)

IN Horrobin, David Frederick, Guildford, United Kingdom|

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SUMM . . . increased expression of E-cadherins and inhibition of proteolytic enzymes such as urokinases, lipoxxygenase and matrix metalloproteinases, and inhibition of cancer-associated cachexia

SUMM (s) cancer cachexia.

. and prostatitis; impotence and male infertility; mastalgia; male pattern baldness; osteoporosis; dermatological disorders; dyslexia and other learning disabilities; and cancer cachexia; comprising administering to a patient in need thereof an effective amount of the compound of claim 10.

. disorders; cardiovascular disorders; degenerative diseases of the eye; psychiatric disorders; osteoporosis; dermatological disorders; dyslexia and other learning disabilities; and cancer cachexia; and wherein R.sup.1 is eicosapentaenoic acid (EPA) and R.sub.2 is selected from the group consisting of eicosapentaenoic acid (EPA) and.

64. A method for treating cancer cachexia, comprising administering to a patient in need thereof an effective amount of the compound of claim 10.

IT 53-86-1, Indomethacin 88-82-4, 2,3,5-Triiodobenzoic acid 506-26-3
1077-28-7, 1,2-Dithiolane-3-pentanoic acid 10417-94-4
54562-14-0 98770-65-1

(preparation of fatty acid esters as bioactive compds.)

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4. End with /Q for a query (search profile, structure, or screen set), /A for an answer set, or /L for an L-number list.
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6. Not be END, SAV, SAVE, SAVED
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